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Mass. Dept. of mental diseases

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OF THE

MASSACHUSETTS COMMISSION

ON

MENTAL DISEASES.

(PUBLISHED QUARTERLY BY THE COMMISSION.)

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SELECTED MEDICAL AND SCIENTIFIC STUDIES.

A KEY TO THE PRACTICAL GROUPING OF MENTAL DISEASES.*

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Abstract.

Steps in the logical classification of disease.
The task of the psychiatrist contrasted with the alienist's.
Insanity not an entity; neither are all mental diseases *sui generis*.
Disease-group diagnosis in psychiatry, analogous to botanical classification of "orders."
Ten disease-groups or "orders" plus an eleventh for the residuum of cases.
-osis and *-aceæ; Rosa gallica* and *Neurosyphilis paretica*.
The key principle.
Special discussion of the eleven groups, syphilitic, feeble-minded, epileptic, alcoholic, drug and poison, focal brain (including traumatic and arteriosclerotic), somatic, senescent-senile, dementia præcox, manic-depressive, psychoneurotic and psychopathic.
Conclusions.

Key to the Practical Grouping of Mental Diseases.

The layman makes out in the great majority of cases the gross fact of sickness. The layman works as it were with the naked eye of mere observation. The practitioner soon replaces the gross "*looks sick!*" with the more concrete "*looks surgical,*" "*contagious,*" "*obstetric,*" "*mental,*" "*nervous,*" etc. The general practitioner advances from the unarmed eye of mere observation to the lens-like power of trained comparison. He is termed "general" in that he deals with great groups, classes or orders of diseases. But these great groups fall short of the true aim of diagnosis, — the pragmatic aim of showing what is best to do. These group captions indicate general lines of treatment, corresponding with but the lowest power of the logical microscope. Resort must be had to the specialist, if such exists and is available (or the general practitioner must be his own specialist); in short, a higher power of the logical microscope is invoked and "*surgical,*" "*contagious,*" etc., get resolved into "*abdominal,*" etc., "*exanthemata,*" etc. Finally still higher powers of the logical microscope, "oil-immersion systems," as it were, are applied, and the so-called entities, the latest constituents of the nosology of the day, roll into view. Thus, with the progress from mere observation to comparison and synthesis, "*looks sick*" turns, *e.g.*, into "*mental,*" and "*mental*" turns, *e.g.*, into "*syphilitic,*" and "*syphilitic*" turns, *e.g.*, into "*general paresis.*"

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* Read at the forty-third annual meeting of the American Neurological Association, May 21, 22 and 23, 1917. Reprinted from The Journal of Nervous and Mental Disease, Vol. 47, No. 1, January, 1918.

In the uneven progress of diagnostic science, as roughly marked out by medical-school departments and by the great specialized medical association, the "mental" has not been the most favored of the great groups. Alienists, however, are slowly but surely being replaced with psychiatrists. Alienists are required by the exigencies of legal medicine to make the generic distinction "mental," *i.e.*, "insane," and the more canny of the alienists stop with this distinction. Psychiatrists are those whose duty requires them to go farther and identify groups of mental diseases and the entities themselves. The task of the psychiatrist is super-legal, special, concrete, not legal, general and abstract like that of the alienist. When alienists and psychiatrists are brought into the same courtroom, as in the Thaw case of evil memory, havoc is apt to be played both with legal and with medical distinctions.

In presenting this key to the grouping of mental diseases I want to make clear exactly what stage of classification I am referring to. (1) The layman is supposed to have made his gross observations. (2) The general practitioner is supposed to have placed the case in the great group of the mental. As yet no alienist is supposed to have approached the case, endeavoring to determine "insanity" for the benefit of some court and to satisfy the dictates of some code of lunacy laws. No! the next phase of inquiry is that of (3) the psychiatrist or of the general practitioner specializing as a psychiatrist.

The object of this psychiatric inquiry is, not to determine what would satisfy a court as to committability, but to determine from a physician's point of view what the disease in question is.

The alienist may enter, if he likes or if it is necessary, to determine the medically irrelevant fact of insanity. We shall be even willing to supply the alienist with pertinent records and observations. It is even sometimes true that psychiatrists make reasonably good alienists, just as they may make good hospital superintendents or good laymen. But it must be insisted that the medical problem is distinct from the legal problem, — that the problem of the psychiatrist is not that of the alienist.

The psychiatrist now confronts his "mental" — or, more exactly, his psychopathic — material, freed for the time being of medico-legal (*i.e.*, "committability" or "insanity") considerations. How shall the entity be reached, viz., something to correspond in mental diseases to scarlet fever among the contagious diseases?

The easiest way out, perhaps, is to deny the existence of entities in mental disease. There are two forms of this contention, — first, that mental disease is nothing more nor less than insanity, an entity itself, a genus with but one species; secondly, that all victims of mental disease are individually to be provided with entities, that is, all examples of mental disease are *sui generis*. The development of psychiatry has killed the former contention stone-dead, but the latter contention still flourishes to an extent among those who overstress the "individual factor." And this latter contention is bolstered up by the existence of so many psychopathic patients of whom a diagnosis cannot be rendered for practical or theoretical reasons. However, there are no really consistent advocates of the *sui generis* plan of classification.

Following is my own plan for the analytical consideration of available data in a case of mental disease. I achieve thereby a provisional diagnosis or group-diagnosis which I regard as an important step towards final diagnosis, *i.e.*, diagnosis of the particular group-member or entity. It is hardly necessary to interpolate that if I can achieve the entity by skipping the stage of provisional diagnosis I shall do so cheerfully. This list is not a list merely, but a sequence, a key to be followed, when necessary, like a botanical key in the search for the classification of a plant.

I have stressed the key-principle in the method of grouping adopted, and shall linger a little on the botanical analogues of the key and the ordered group-list. Omitting details (discussed *infra*), the sequence of mental disease-groups I have provisionally arranged as follows: —

Mental Disease Groups (*Orders*).

1. Syphilitic,	Syphilopsychoses.
2. Feeble-minded,	Hypophrenoses.
3. Epileptic,	Epileptoses.
4. Alcoholic, drug, poison,	Pharmacopsychoses.
5. Focal brain ("organic," arteriosclerotic),	Encephalopsychoses.
6. Bodily disease ("symptomatic"),	Somatopsychoses.
7. Senescent, senile,	Geriopsychoses.
8. Dementia præcox, paraphrenic,	Schizophrenoses.
9. Manic-depressive, cyclothymic,	Cyclothymoses.
10. Hysteric, psycho-, neurasthenic,	Psychoneuroses.
11. Psychopathic, paranoiac, *et al.*,	Psychopathoses.

Disregarding for the moment both the order and the sufficiency of the list let us consider the nature of the terms. The ending *-osis* was chosen for these groups to correspond exactly with the ending *-aceæ* for certain botanical groups, the so-called orders of plants (for simplicity's sake I pursue here only the botanical, not the zoölogical analogy). The parallel can be shown thus: —

Rosaceæ,	order,	Syphilopsychoses.
Rosa,	genus,	Neurosyphilis.
gallica,	species,	paretica.
R. gallica,	name,	N. paretica.
red rose,	common name,	general paresis.

Or: —

Cucurbitaceæ,	order,	Hypophrenoses.
Cucurbita,	genus,	Hypophrenia.
Pepo,	species,	idiotica.
C. pepo,	name,	H. idiotica.
pumpkin,	common name,	idiocy.

Or: —

Rubiaceæ,	order,	Epileptoses.
Coffea,	genus,	Epilepsia.
Arabica,	species,	tarda.
C. arabica,	name,	E. tarda.
coffee,	common name,	late epilepsy.

From this hint it may be seen that the use of the plan does not necessarily contemplate renaming diseases, although this may be often very desirable. It is not that a sufficient diagnosis of a given disease is, *e.g.*, "syphilopsychosis," any more than it would be proper to stop in plant diagnosis with *Ranunculaceæ* when the true goal was *Hydrastis canadensis.*

Whereas the ending *-osis* suitably expresses ordinal characters, the ending *-ia* (as in epilepsia, neurasthenia) suitably indicates generic characters of disease; indeed some dictionaries regard *-ia* as primarily suggesting disease. The ordinal ending here preferred is accordingly *-osis;* the generic ending *-ia.*

So much *re* nomenclature may suffice at this time (see below under the separate groups for further remarks). Aside from certain novelties in nomenclature the plan has, I believe, merit as an application of a key-principle. Even if one were content with ordinary English group names ("syphilitic," "feeble-minded," etc.), still the order of consideration of group-data would remain of commanding importance. Should not a shred remain of the nomenclature just presented, the key-principle in the analysis of clinical psychiatric data would remain of use.

I commend the analysis of clinical psychiatric data upon a key-principle, much as by trial and error in the botanical process of classification. I do not, on the other hand, especially commend the gathering of the data in this order or in any special order. After the data are collected by some impartial process of observation and regular process of recording, I would then proceed to the analysis of the data in the order mentioned. I would give every hypothesis the completest opportunity to verify itself. I would even impress slight symptoms and minor indications into the service of each hypothesis in succession. This artificial bias is perfectly safe, since I am going down the line of possibilities giving the same bias in succession to each. For there is one striking difference between botanical classification and medical diagnosis. In botanical identification (as with Gray's botany) if I reach *Hydrastis canadensis* I hardly need to go farther, so rare are true plant hybrids. But in medicine a man may be victim of several diseases, and one must consider all pertinent possibilities (see below for remarks on "combined psychoses").

The whole matter of quasi botanical and zoölogical classifications in the psychiatric field would gain from a review on modern lines of the older classifications from de Sauvages and Linnæus; but this is not the place for an elaborate review.

Without further introduction I pass to remarks on the eleven groups themselves. I purposely omit consideration of the disease "genera" under these "orders," because there must obviously be so much controversial in most groups; for the data are not all gathered for a proper taxonomy! However, under the syphilitic and feeble-minded groups I have made suggestions for the "generic" subdivision of these "orders."

1. *Syphilopsychoses* (*the Syphilitic Group*). — The main representatives of this group are general paresis (otherwise general paralysis of the insane, paralytic dementia, "softening of the brain") and the so-called "cerebral syphilis," preferably termed cerebro-spinal syphilis. I have recently tried to bring more order into the nomenclature by throwing together all diseases of the nervous system traceable to syphilis under the term neurosyphilis, the main forms of which are (*a*) paretic, (*b*) tabetic, (*c*) diffuse, (*d*) vascular, (*e*) juvenile and (*f*) gummatous. The majority of these forms of neurosyphilis are at one or other period characterized by mental symptoms, so that the six-heading neuropathological classification works well enough for the psychoses more narrowly taken. However, the tabetic psychosis (a variety of *Neurosyphilis tabetica*) is exceedingly rare, so rare that I question my ever having seen an instance. On the other hand, mental disease supervening in tabetics is of course common enough, and will ordinarily turn out to be part and parcel of general paresis (*Neurosyphilis paretica vel taboparetica*), though sometimes the symptoms will prove due to intercurrent syphilitic blood-vessel disease (*Neurosyphilis arteriosclerotica* plus *Neurosyphilis tabetica*). It is clear that examples will be found of *Neurosyphilis diffusa*, *i.e.*, of a mental disease depending on a widely diffused process, possibly involving meninges, parenchyma and vessels as well, but having a prognosis far more favorable *quoad vitam* than *Neurosyphilis paretica*. Where possible, the diagnosis should certainly seek to pick out the meningeal, parenchymatous and vascular features, or their combinations, and distinguish these factors in the designations chosen. Something like this has been attempted by Head and Fearnsides, although the *syphilis centralis* of these authors seems not too happy a term, permitting the diagnostician to escape without a prognostic comment and without specifying what he really thinks is going on in the "central" substance.*

2. *Hypophrenoses* (*the Feeble-minded Group*). — The main representatives of this group are the well-known graded forms of feeble-mindedness (as we use the term generically in America),

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* For full discussion see Southard and Solomon, "Neurosyphilis: Modern Systematic Diagnosis and Treatment presented in 137 Case Histories" (Monograph Number Two of the Psychopathic Hospital, Boston), Boston, W. M. Leonard, 1917.

viz., (*a*) idiocy, (*b*) imbecility and (*c*) feeble-mindedness proper (the morons of Goddard, collected by Tredgold under the term *morosis,* in point of fact, a term used by Linnæus). The excellent results of modern mental testing, however, have left us with a fourth group of (*a*) subnormals, lying between the morons below and the normals above; these may be known as dullards, simples or stupids (Tredgold), among whom none should be included save .those who actually *measure* low by modern scales and gradings. Thus, above (*a*) *Hypophrenia idiotica,* (*b*) *H. imbecilla* and (*c*) *H. morotica* we should find (*d*) *Hypophrenia subnormalis* (*sc. metrica*). But, as practical work abundantly shows, there are other hypophrenoses in which available measurements leave us at loose ends or at a loss. These Tredgold inclines to term *Amoralia* on account of their moral deficiencies and as a condensed term for Prichard's moral insanity (or a part thereof). Perhaps the reference to conventional standards implied in such terms as moral insanity or imbecility, or amoralia, is more or less justifiably resented by modern workers. Moreover, sometimes the deficiency is not in morals and yet exists. "Congenital psychopathic inferiority" is a term which covers many of these cases, being perhaps a rough translation of *Minderwerthigkeit.* "Below par" we sometimes say of these cases, being a figurative usage of the term "par" when no method exists of determining what is par (*Minderwerthigkeit* has the same air of greater exactitude than exists). On the whole perhaps we do less violence to existing terminology if we speak of (*e*) *Hypophrenia amoralis.* I often think of the group as one of non-metric or qualitative feeble-mindedness, though it cannot be denied that "qualitative weakness" is a contradiction in terms. This latter objection would be the stronger if we were trying to describe a scale of intensity in some one attribute. In many instances, it has seemed to me as a psychiatrist, these cases of qualitative inferiority are marked, not so much by a weakening of faculties as by a total absence of various faculties, — faculties, of course, that are not indispensable in life. Without endeavoring to prove or expound the idea, what would happen could the instinct of disgust be conceivably left out of a subject's makeup? One could not then measure it; one could merely find it lacking. High evolutionary complications, *e.g.*, disgust, have simply not entered the subject's make-up. Accordingly, I personally would prefer (*e*) *H. simplex* (*sc. nonmetrica, qualitativa*) to *H. amoralis.*

The classification might go —

(*a*) *H. idiotica.*
(*b*) *H. imbecilla.*
(*c*) *H. morotica.*
(*d*) *H. stupida vel subnormalis* (*sc. metrica*).
(*e*) *H.** *simplex vel amoralis* (*sc. non-metrica, qualitativa*).

I may be pardoned a word on behalf of the feeble-minded patients whom we now so often demonstrate in clinics; it has proved of value to use terms which do not possess unfortunate connotations for the minds of these patients. Of course, idiots are not harmed by hearing the term, and the imbeciles not to any extent. It is all very well to call a spade a spade; but if the listener transforms a "spade" into a pile-driver in his own mind, Richard Cabot's warnings about belligerent truth-speaking fall into queer cross-lights. I find it of value to use in clinical demonstrations perfectly exact terms which the patients have no chance to misconstrue. *Hypophrenia morotica, H. subnormalis, H. simplex* are such terms.

As to the term *Hypophrenosis* itself, it has been suggested to me that *Hypopsychosis* would be preferable. Perhaps so. I considered that "phren" had been used by Kræpelin in his proposal of *Oligophrenia* for feeble-mindedness. Oligophrenosis was then the natural group rendering for the "oligophrenias." I could not see that it was well to preserve the connotation "few" in the prefix "oligo-," which erroneously suggests "*few*-mindedness" where inferiority is really in question. But the other half of Kræpelin's suggestion seemed good, in view of the intellectual trend of the Greek term "phren," which closely imitates the intellectual trend of the term "mind." The "psyche" seems somehow broader than the "phren," just as "soul" seems broader than "mind," at least when both are used. Accordingly, hypophrenosis seemed to be the nearest euphonious Greek equivalent of the term "feeble-mindedness," a term which, lying in the midst of the group, has succeeded in dominating the whole.

3. *Epileptoses* (*the Epileptic Group*). — Without here entering the vexed question of the classification of the epilepsies themselves, I will use the heading to call attention to a feature in the usage of the key to classification of which this group forms a part.

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* Or should there be a new genus? *Amoralia Prichardi?*

If a disease be syphilitic, *i.e.*, if syphilis played an essential part in its causation and is the practically most important factor in that causation, then feeble-mindedness, epilepsy, coarse brain disease and the rest remain subordinate. Whether a disease be *Neurosyphilis hypophrenica* (as a rule, of course, *idiotica* or *imbecilla*) or, on the other hand, *Hypophrenia syphilitica* may be a matter of discussion; practically, however, it is my present contention that such cases be placed in the syphilitic group, really because of our bounden therapeutic duties to these cases.

The same holds for the epilepsies, and so down the line. "Syphilitic epilepsy" is a term good from the epileptologist's point of view; from the point of view of the physician and therapeutist in general it is better to classify the case as one of "epileptic syphilis," viz., in our nomenclature, *Neurosyphilis epileptica.*

4. *Pharmacopsychoses* (*Alcohol, Drug and Poison Group*). — This designation (like hypophrenoses for the feeble-minded group) is a novel term, but one of such obvious fitness for a well-recognized group that I can conceive no reason against its adoption, if the group principle itself is deemed worthy.

Without entering the familiar field of the alcoholic psychoses (delirium tremens, alcoholic hallucinosis, jealousy-psychosis, dementia, etc.), and without discussing morphinism, etc., I will take occasion to point out how in the successive consideration of data pointing in a given case to (1) syphilis, (2) feeble-mindedness, (3) epilepsy, (4) alcohol and drugs we are able to collect all the data that would be of commanding value in immediate treatment in special institutions. To be sure, special institutions for neurosyphilis have not so far been established in the world, though there is much to be said for the plan.* But aside from neurosyphilis or "salvarsan" hospitals, advanced communities will always be found providing special institutions for the feeble-minded and the epileptic. It is an established principle of mental hygiene that the feeble-minded and the epileptic shall be treated separately. Placing a case among the hypophrenias or the epilepsias means, as a rule, something wholly practical; it means that special institutional treatment is indicated (*e.g.*, in Massachusetts, "this is a Waverley case" or "this is a Monson case;" in New York, "this is a case for Rome" or "for Sonyea"); or, if the actual State institution is contra-indicated, then something equivalent or better along the same line is prescribed. As to the pharmacopsychoses, very few States have proceeded to the experiment of special institutions, though Massachusetts, for example, contains a successful one for moral and physical uplift cases. The "rounder" group and the delirium tremens problem are, so far as I am aware, not adequately dealt with on the planet. Not to launch into a homily on mental hygiene, let me merely insist that by considering, eliminating or appreciating the data of these four groups we have dealt with some of the largest social problems in psychiatry. How often in the elaborate discussion of doubtful mental cases do we find our younger officers indulging in long, not to say acrimonious, discussions of the relative merits of dementia præcox and manic-depressive psychosis, only to learn at the end that the case was essentially syphilitic, feeble-minded, epileptic or alcoholic! And how often do we hear circular controversies about the respective parts played by epilepsy and alcohol, or by alcohol and dementia præcox in certain cases! The practical diagnosis is brought out at once, as a rule, by the simple inquiry, "Do you desire this case *treated* as an epileptic primarily, or primarily as an alcoholic?" or, "Do you fancy this case in a hospital for dipsomaniacs or do you want him placed in a hospital for the insane?" It is especially in this group of pharmacopsychoses that these pragmatic questions acquire their greatest significance.

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* The Massachusetts Commission on Mental Diseases and the Grafton State Hospital Board have co-operated in the establishment of a neurosyphilis ward, operated by special investigators.

5. *Encephalopsychoses (the Focal Brain Group, including Arteriosclerotic, Tumor, Traumatic Cases, etc.).* — This is the "neurologist's group" of mental diseases. The classification is of course not as broad as that of nervous diseases as a whole, because there are still many nervous diseases without a characteristic psychotic side. Still, in general, the neurologist's technique in virtually its entirety is required in the elucidation of the encephalopsychoses. The neurologist is of value elsewhere, notably in the syphilitic group, precisely because many of the syphilopsychoses are attended by coarse brain disease or by spinal cord disease deranging brain functions. Here, then, is the group in which the condition of the reflexes, the sensations, the motor and secretory powers, as determined by the neurologist, is of decisive worth in diagnosis.

The practical worker may be astonished at one feature of this group, seemingly so heterogeneous. It is the habit — a lax one, I believe — of many workers to unite in their minds too closely the arteriosclerotic and the senile psychoses. I place the arteriosclerotic cases in the encephalic (focal brain) group, because I believe that in the present phase of psychiatric science the diagnosis "arteriosclerotic psychosis" cannot be safely made except in the presence of some of the neurologist's signs, or supported by shocks or other seizures in the anamnesis. I do not here speak of the mental sequelæ of somatic arteriosclerosis or of generalized arteriosclerosis; the arteriosclerotic subgroup of the encephalopsychoses is a group, according to this interpretation, of which "organic neurological" symptoms, signs and history are characteristic. Some may miss the opportunity of ascribing "general deterioration" to arteriosclerosis; the present key requires investigation to learn whether the disease in question is assumed to be due to focal brain vascular lesions (encephalopsychoses) or to senility (see below, geriopsychoses) or to generalized arteriosclerosis (somatopsychoses, cardiovascular group) or, of course, to syphilis itself.

I would maintain a somewhat similar practical value for this key in the traumatic field. How many protracted, nay, meaningless, medicolegal discussions could have been dispensed with could the question, *whether the trauma was encephalic structural injury or not,* have been settled forthwith! A case for the present key is placed in the encephalopsychoses, traumatic subgroup, if there is good neurologist's evidence of some sort of focal brain disease; but it is placed far away in the group of the psychoneuroses if it has features predominant of the so-called traumatic neuroses. (It may be superfluous to say that this key does not infallibly place a case in the *right* major group.)

It is only fair to ask whether a line can be drawn between "focal" and "not-focal" brain disease. For instance, sometimes a case with multiple miliary aneurysms of the brain substance comes up for discussion. To be sure, such a case is commonly attended with multiple hemorrhages which would serve to place it forthwith in the focal brain disease-group. Nevertheless, many of the nervous and mental symptoms are doubtless due to lesions that could hardly be seen by the naked eye. The point is not so much what "coarse" lesions are demonstrable post mortem, as it is whether the neurologist with his special logical equipment can make the diagnosis. Or, again, take the brain lesions of certain cases of pernicious anemia. Shall these cases be placed among the encephalopsychoses? The brain lesions are microscopic. But on the criterion of the encephalopsychoses as the neurologist's group, I am inclined to say that these cases should be counted amongst the encephalopsychoses. I believe I should also desire to place the rare Alzheimer's disease here also, on the ground of its "organic," not senile, appearance.

The plot obviously thickens hereabouts. Miliary aneurysms and Alzheimer's focal lesions, however largely invisible to the naked eye, are nevertheless lesions which cut mechanisms apart precisely as larger lesions cut them apart. The lesions are, as it were, globar or molar lesions, and not of the diffuse and intimate character of those in many diseases. With the pernicious-anemia brain one is not so dogmatic. Perhaps, if we knew the somatic origin of pernicious anemia, we should prefer to classify this condition among the somatic psychoses. As for pellagra, I should prefer to place its psychosis in the next group.

6. *Somatopsychoses (the Bodily Disease Group, including Infective and Exhaustive Cases, Cardiac Cases, etc.).* — This is the "internist's group" of mental cases.

The term "soma" is sometimes opposed to "psyche," sometimes to "head," sometimes to "brain." The hylozoist might oppose this term on the ground that all mental diseases are "somatic." An ardent dynamicist might query whether any mental disease is importantly "somatic." An interactionist might object on both grounds.

I am here innocently trying to oppose the term to the "brain" or "encephalon." After eliminating (1) syphilis, (2) feeblemindedness, (3) epilepsy, (4) alcohol and drugs, (5) the "organic neurological" conditions, I then turn to (6) the organic non-nervous field. I apply the internist's technique. I perhaps find some "symptomatic psychoses" that appear to be intimately dependent upon cardiac or renal conditions; or again, upon infectious disease, such as typhoid fever or pneumonia; or again, upon non-nervous tumor, diabetes, metabolic disease, etc. As above mentioned, it would seem well to consider the possibility of pellagra at this point, perhaps also pernicious anemia (provisionally, however, placed among the encephalopsychoses so far as its mental side is concerned).

There, too, may be placed the glandular affections, — hyperthyroidism, myxœdema, cretinism and the like.

7. *Geriopsychoses* (the Senescent-senile Group).* — Both the name and the key-position of the senescent-senile group may perhaps be questioned. As to the key-position, it might be

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* Better, *Presbyopsychoses?*

queried whether it would not be better to exclude dementia præcox and manic-depressive psychosis before invoking involutional processes to explain a given mental disease. Still, in the use of this key, one is not supposed to stop with the first plausible group into which the clinical picture might fit. Accordingly, one would be assumed not to omit a study of the data from these other points of view.

I was moved to place the old-age group seventh, partly to range it with more definitely organic psychoses, and partly because I felt it better not to divide the succession that follows (see groups 8, 9 and 10).

"Geriatrics" is a term that is very properly coming in to cover a definite but ill-developed specialty dealing with old-age disorders. I want to use "geriopsychoses" to cover, not merely the pronounced and ultimate disorders of old age, but the disorders of senescence, the presenium and the involution period. It is, at any rate, practically important to consider all the data together that look in this direction, along the down gradient.

As above stated, I feel that it is practically important to separate the arteriosclerotic group from the senescent-senile. Not that arteriosclerosis is not a frequent phenomenon in old age (as it is assuredly also often not a marked factor). One may truly be as old as one's arteries, but the arteries are not always as old as many other parts of the body. Aside from all discussion as to what constitutes old age, the prognosis of arteriosclerotic psychosis is often quite other than that of senile dementia with which it is so frequently confused. Accordingly, I believe that practical psychiatrists will agree that it is a practical advantage to consider the signs of arteriosclerotic psychoses separately from those of the senescent-senile group.

8. *Schizophrenoses (the Dementia Præcox or Schizophrenic Group).* — As "dementia præcox" forms no proper adjectives, the choice by Bleuler of the term "schizophrenia" for dementia præcox has been welcomed on all hands. Kræpelin himself, although he still speaks of the disease dementia præcox, often uses the adjective schizophrenic for the victims of the disease.

The idea of schizophrenia, viz., "dissociation" or "splitting" of mental processes, lies at the bottom of much, if not all, that is distinctive of the mental-symptomatic side of dementia præcox.

There is therefore a good deal to say for building a group-designation of schizophrenia, the symptom. Kræpelin, to be sure, in his last edition groups dementia præcox and paraphrenia (a renamed French disease, *délire chronique à evolution systematisée*) under the general term "endogenous deteriorations." But this term "endogenous deteriorations" seems far too general, besides which "endogenous" is a term which Germans (following Moebius) have specialized to mean "taking rise within the nervous system," a meaning possibly warranted, but certainly not generally accepted.

Of course, practical workers are apt to use "dementia præcox" for disease which, though functional looking, is regarded as fated to deterioration. Although everybody is theoretically aware that not all cases of dementia præcox deteriorate, yet the "lure of the 100 per cent." and the catchiness of the term "dementia" lead to a practical disregard of the actual guarded prognosis of dementia præcox and a virtual claim that all "real" cases of dementia præcox deteriorate. One is familiar with clinics in which the diagnosis of functional-looking diseases is from time to time revised in favor of dementia præcox if deterioration has set in, and in favor of manic-depressive psychosis if there has been no deterioration. The fact that Kræpelin observed that dementia præcox patients are apt to deteriorate *in particular and characteristic ways* is revised by practical men to the effect that dementia præcox deteriorates, and, if not, the diagnosis is wrong.

In a clinic for doubtful cases, like that of the Psychopathic Hospital in Boston, I find that the diagnoses tend to vary somewhat with the natural pessimism or optimism of the workers. I find it practically of value to limit the diagnosis dementia præcox to cases in which something schizophrenic can be found. We may thus score too low a tally of correct diagnoses of dementia præcox, but I am sure we save some cases from an unjustifiably pessimistic diagnosis, for at best the prognosis of the schizophrenias is not a good one.

9. *Cyclothymoses* (*Manic-depressive Group*). — Just as "dementia præcox" forms no proper adjective, so we find the same difficulty with "manic-depressive psychosis." No one has ever been satisfied with the term "manic-depressive psychosis," although the Kræpelinian conception of the disease which goes by that name has been pretty generally accepted.

The term cyclothymia has been used by some authors for all or part of the disease-group known as manic-depressive psychosis. The cyclic nature of the course of many if not the majority of instances of this psychosis is embodied in the first part, cyclo-, of the term "cyclothymia." The most prominent feature, psychologically speaking, of manic-depressive psychosis is the emotional disorder, which is demonstrated either in the morbid gaiety, fear, anger or depression which characterizes different phases of the disease. Even those complex examples of the disease in which mania and depression occur mixed (cases, as one might say, of "mixed emotions") are still fundamentally examples of emotional rather than intellectual disorder.

Some authors have applied the term "affective" to the group which may be known as the group of the affective psychoses.

The second theme, thym, of the term "cyclothymia" refers to this predominantly emotional or affective nucleus of the disease or diseases known as manic-depressive psychosis. Kræpelin himself occasionally uses the term cyclothymic. Accordingly, I have felt that if we desire to place in a single great group the varieties of manic-depressive psychosis and allied diseases we could do no better than to speak of them as the cyclothymias, or, adopting the common Greek ending for these groups, the cyclothymoses.

It will be noted that the schizophrenias have been placed in this key prior to the cyclothymias. Practical workers will all recognize how easy it is to find "cyclothymic" (that is, "manic-depressive") phenomena in all sorts of mental diseases and conditions which are not genuinely manic-depressive. These cyclothymic symptoms are even not infrequently found in schizophrenic cases. In diagnostic discussions where numerous physicians are consulting, not infrequently one physician is found triumphantly proclaiming dementia præcox on the basis of schizophrenic symptoms, whereas his colleague with equal triumph claims manic-depressive psychosis on the basis of cyclothymic symptoms.

The point of the group arrangement in this key is perhaps nowhere more clearly shown than in the priority that the schizophrenias take as against the cyclothymias. In practice it seems that schizophrenic phenomena blanket cyclothymic phenomena. A victim of dementia præcox may in several parts of his mental life exhibit perfectly good and clear-cut cyclothymic phenomena, yet it is the schizophrenic or dissociation phenomena which, according to this point of view, must command the diagnosis. This situation can the more readily be understood when it is remembered that, after all, the majority of the manic-depressive or cyclothymic phenomena are merely exaggerations or diminutions of normal functions. After all, there is nothing "queer" or dissociative, nothing that definitely proves mental disease in the supernormal gaiety or exaggerated depression of a manic-depressive subject. Perfectly normal subjects may at times show these features, though perhaps not to the extreme extent nor in the rich display that characterizes the manic-depressive patient.

The victim of dementia præcox, according to our point of view as above expressed, must have something in his status or history to show schizophrenia; but elsewhere in his status or history he may very well show those variations above and below the normal in emotions which normal people, as well as manic-depressives and other psychopaths, show. In short, the cyclothymic patient shows variations of emotion which are decidedly within the frame of the normal, and can rather readily be understood by the layman.

The manic-depressive is either supernormal or subnormal in his emotional display at a given time, and so far as the layman can tell is often doing only what a normal person might do under the circumstances. The concept of schizophrenia, on the other hand, is not one which the layman readily grasps; it is, in fact, fundamentally hard to define.

10. *Psychoneuroses (the Group of Neurasthenia, Psychasthenia and Hysteria).* — It may seem that the psychoneuroses should not be grouped among mental diseases. One suspects, however, that the authority who takes this line is under the influence of older notions as to the supposed identity of mental disease with certifiable insanity.

From the standpoint of general practice, or of clinic which lies nearest to general practice, namely, the psychopathic hospital clinic, the psychoneuroses are precisely as much within the frame of psychiatry as the schizophrenias and cyclothymias.

As far as the gravity of these conditions is concerned, there is not much doubt that many of the psychoneuroses are just as grave in prognosis as many of the cyclothymias. It may be suspected that some of the cyclothymias are in practice termed psychoneuroses precisely because the diagnostician fears that the patient and his friends will resent the diagnosis manic-depressive psychosis, when the diagnosis neurasthenia or psychasthenia will not be resented or feared.

It cannot be denied, however, that the demarcation of the psychoneuroses is an extremely important one from the practical standpoint of prognosis. Whereas the schizophrenic group takes its own way, and whereas the cyclothymic diseases seem in a sense self-limited, or at all events beyond the reach of special therapeutics, on the other hand the psychoneurotic group is the psychotherapeutic group par excellence. We practically mean when we call a case psychoneurotic that we feel that the case has a good chance under methods of direct or indirect psychotherapy.

It might be inquired whether the psychoneuroses should not be placed nearer the head of our key. Errors in diagnosis are undoubtedly found in which some other form of mental disease is asserted to exist when psychoneurosis is the fact. However, we have come upon so many instances in which syphilis, alcoholism, arteriosclerotic brain disease, somatic disease and manic-depressive psychosis have been erroneously regarded as psychoneurotic that I feel confident that these conditions should be considered and eliminated prior to making the diagnosis of neurasthenia, psychasthenia or hysteria. Of course, if a wrong diagnosis of some other form of mental disease is rendered, then it is conceivable that the psychoneurotic patient will fail to get what he so importantly needs, namely, psychotherapy. However, the will to psychotherapy is so deeply implanted in every physician that I feel few cases have genuinely suffered from the lack of an attempt to carry out psychotherapy. Of course, no one would claim that every psychoneurotic gets the proper kind of psychotherapy or enough of it; but that is another story.

11. *Psychopathoses* (*the Group of the Psychopathias and of a Variety of Doubtful Entities*). — I shall not here discuss at length this doubtful group. I place therein a variety of ill-defined conditions. Some of these, as the psychopathic personalities, verge very closely upon the normal; others, like the so-called prison-psychoses, are considered by some authorities not to exist at all, at least as entities.

The true nature of the so-called "psychosis of the deaf" is not well enough understood to permit its characterization; at all events, it does not appear to fall among the ten well-defined groups listed above.

Those rare cases of true paranoia I personally prefer to place in this ill-defined group of psychopathoses. At any rate, I see no special advantage in making these pure paranoias, without signs of mental dissociation or tendency to deterioration, tag along behind the schizophrenias.

I would also place in this ill-defined group those rare cases of mental disease which are regarded as certifiable and deserving of institutional régime, but which cannot be practically classified in any one of the above-mentioned ten groups.

Of course, in practice these "undiagnosticated insanities" are really the fruit, as a rule, of poor observation or of poor opportunity for observation.

At this point it may be inquired whether such entities as combined psychoses need be assumed. For my part, I am wholly willing to agree that a patient might properly enough be theoretically placed in any one of several of the groups above mentioned. Thus a patient might well be an old man with coarse brain disease and disease elsewhere in his soma who had acquired syphilis, was extremely alcoholic and had been a feeble-minded subject to start with; and this subject might, of course, have shown a variety of psychoneurotic and cyclothymic symptoms. It is even possible that such a patient might exhibit some phenomena that looked like those of a dementia præcox. Practically, however, such a case would be placed in one of the above-mentioned ten groups precisely because the diagnostician would want to have the case treated primarily either as a syphilitic or as a feeble-minded subject or as an epileptic, etc. Accordingly, we may provide full scope for the so-called combination of psychoses in theory without damaging the practical validity of the above-mentioned classification.

SUMMARY AND CONCLUSIONS.

I have here presented not so much a classification as a key to the grouping of mental diseases. The key has been worked out to the extent of ten well-defined groups and an eleventh residual group. These groups correspond to the groups of, *e.g.*, the *Rosaceæ* or *Leguminosæ* of botany, and do not correspond to the genera and species of those orders. Some hint is given of the generic and specific distinctions of mental disease that might correspond to the genera and species of botany, provided that there were any practical need for a quasi-botanical or zoölogical genus-species distinction in mental diseases.

The incentive to this grouping has been practical. No endeavor was made on the library table to construct a *hortus siccus* of mental diseases. On the contrary, this key is the product of several years of work in the Psychopathic Hospital in Boston, where the task of reasonably accurate diagnosis by an ever-changing staff of psychiatrists-in-training was the desideratum.

I do not accordingly suggest this key as something to replace the methods of the expert in arriving at a conclusion concerning psychiatric diagnosis. I do offer it, however, as a guide for the tyro and the psychiatrist-in-training. It is not an outline giving an order of examination. It is a scheme for summarizing and evaluating results after the physical, mental and historical data are collected. The plan is eliminative, but is subject to this reservation, — if one arrives in the chosen sequence of analysis at a plausible or even a correct group diagnosis one is not thereby absolved from continuing the process of analysis. All data bearing on any of the groups must be considered. Diseases may be "hybrid," though practically one is almost never in doubt as to the group under which to subsume a case. Theoretically, one may be, for example, both epileptic and alcoholic; practically one is either an epileptic alcoholic or an alcoholic epileptic. The guide to the grouping here is a pragmatic one, and depends upon the institution or the special treatment to which the supposed victim of epilepsy and alcoholism must gravitate. I must especially emphasize that the groups and the group names do not correspond to nosological entities and entity names. The placing of a case in one of these eleven groups is not psychiatric diagnosis in the entitative sense. Accordingly, this grouping does not run into collision with any previous endeavor to classify the genera and species of mental disease, such, for example, as the genera and species in the majority of classifications quoted in Hosack.[1]

I would insist, further, that the group headings given are not special enough to constitute sufficient diagnosis for a classification of use in the statistics of institutions for the insane. The plan is not so much an excursion in nosology as an essay in the technique of psychiatric diagnosis for the tyro. The plan gives hints for a method of arriving at an eventual diagnosis; it does not prescribe the names of diseases. Again, the plan is not an etiological plan, although recent advances in psychiatric etiology have been such that many of the practical groups are actually etiological groups.

It is possible that the sequence has been unduly telescoped. It is possible that there should be a traumatic and an arteriosclerotic group. I have placed both of these groups in the encephalopathic or coarse brain, or "neurologist's" group, feeling that I do the diagnostic tyro a service by pulling the encephalotraumatic psychoses far apart from the traumatic psychoneuroses on the one hand, and the arteriosclerotic psychoses far apart from the senile psychoses on the other hand.

Lastly, I would insist once more that the plan is one born of Psychopathic Hospital experience and bred in the first place for the inexpert. It is a key to study and not an analytical classification with any pretense to finality. Elements in the sequence can be destroyed and new elements inserted. Indeed, such processes of extrapolation and interpolation must needs occur in the progress of practical diagnosis. Whatever novelty the plan may have lodges in the sequential character of the analysis of data already collected, and not in the completeness or ultimacy of the groups. The sequential plan of analysis is of course as old as the diagnostic hills. It is superior, however, to the type-matching method of diagnosis in vogue with many tyros, who very often come to their superiors with the plaint that the data in a given case fit the book descriptions of half a dozen diseases. A set sequential analysis of collected data must be superior to a hit-or-miss type-matching of entities.

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1. Hosack, David: "A System of Practical Nosology: to which is prefixed A Synopsis of the Systems of Sauvages, Linnæus, Vogel, Sagar, Macbride, Cullen, Darwin, Crichton, Pinel, Parr, Swediaur, Young, and Good, with References to the Best Authors on each Disease," 1st edition, 1819, 2d edition, 1821, New York.

SOCIAL WORK AS WAR SERVICE.

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Every woman to-day must be asking herself what part she is to take in the war. Exempt from draft, women have a stronger moral obligation to find their proper places, to volunteer where they are needed, or to prepare themselves to volunteer where they will be needed. The greatest burdens of the war will probably not be upon this country for a year or two years, so that there is an amazing opportunity for a woman to fit herself for some kind of work that the world will need. Great as the demand for skilled workers of all kinds is now, there will be a still greater demand a year later, and the interval may serve for acquiring skill in some branch of work that must inevitably be in demand. Insistence upon trained work is the keynote of our national war program.

In social work the scarcity of trained workers is appalling. Our social organizations are now undermanned, and positions are being created faster than workers are being trained. The Intercollegiate Bureau of Occupations states that "for many months there has been a much larger number of positions registered than could be filled." The organizations meanwhile are finding that it is better not to attempt some kinds of work than to employ unqualified persons. This situation is not realized by the disinterested public. The common idea of social work is that it means being kind and helpful to the distressed, a sort of enlargement of the friendly services that all sympathetic and intelligent persons extend to their neighbors; and therefore it is supposed that kind and sensible persons will always be available in large numbers for any social work there is to be done. The public has not yet grasped the fact that social work has become an organized branch of professional activity requiring workers with special qualifications and a definite training, which is offered in professional schools and in graduate departments of universities.

In connection with the war there must inevitably be a demand for social workers that will overwhelm an already inadequate supply. Trained workers will be required for civilian relief; recreational activities in connection with camps; and after-care of soldiers physically or mentally disabled, — blind, deaf, crippled or suffering from nervous disorders. To meet this demand a large corps of women should be under training during the coming year. Approximately full time for a year is the minimum time for preparation for responsible positions. Short courses in stenography, telegraphy, or attendant nursing may seem more alluring. Short courses are being offered in various places to train social workers' aides, and are of value, but should not be confused with the thorough training of the professional social worker. The Canadian Patriotic Fund in Montreal has been remarkably successful in employing untrained volunteer workers, having 700 engaged at one time; yet Miss Reid, director of the Women's Auxiliary, after paying enthusiastic tribute to the spirit and accomplishment of these volunteers, says, "With the numerous opportunities for volunteers in the United States to prepare themselves now under trained workers, many of the problems resulting from inexperience and assuming threatening proportions at times in the Montreal staff should not arise in the course of your work." *

Until recently social work has not attracted college women to any great extent. The social worker is frequently asked by her able-minded friends, "Why do you think it is worth while?" "Do you get anywhere?" "What does it amount to, pulling some people out of trouble while others are falling in?" On the other hand, her more emotional friends will say, "How happy you must be, doing so much good!" "What a wonderful thing it must be to be able to help people!" Both of these points of view have at last met in social work, presumably to become in time firmly bound together in a science whose practice is an art. The fact that as a science social work is in the earliest stages of development will be an incentive to many to take part in its advancement. The literature of social work is scanty, and deals largely with special activities. The philosophy underlying the great aggregation of activities that are generally known under the term "social work" has not yet been presented in any single volume. It is difficult for any one who seeks to know exactly what social work as a profession means, to find out. Observation leads to a bewildering number of contradictions; the literature is confusing and not stimulating to the imagination; the social workers you may talk with are apt to be absorbed in their special job and unready to philosophize. The best hope of under-

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* "War Relief in Canada," by Helen R. Y. Reid, Director and Convener, Ladies' Auxiliary of the Canadian Patriotic Fund, Montreal. Address before the National Conference of Social Work, 1917.

standing the significance of social work is to look about at the signs of the times, — the rapid spread of social service in hospitals throughout the country in the last ten years, the growth of probation systems, the development of parole departments in prisons, the increase in the number of factory welfare workers, the demand for school visitors, the progress of the school center movement. This is the day of individualization, and wherever the problem of relating the individual successfully to his environment arises, there the social worker is needed. As the conditions of the environment improve, it may be expected that the number of individuals requiring assistance in adjustment will decrease; but who can look ahead so far as to see the time when all individuals will be born with normal capacity into a normal environment? Even so the chances of accident would remain, so that the objection often heard, "If society were what it should be we should not need social workers," is a thoughtless misconception. Perhaps the most significant of the signs of the times is the rapid growth of instruction in social work in colleges, both in graduate and undergraduate courses, and the establishment of professional schools for training social workers. A list of the best known of these is appended. A statement such as is made by Dr. E. E. Southard, director of the Psychopathic Hospital, in a talk before the Radcliffe Union, on "Training for Social Work," will still cause astonishment to many whose conception of social work has not advanced with the times, but will indicate to others the certain trend of its development: —

> I would then advocate for prospective social workers a bachelor's degree (with possibly a master's degree in applied work), and I would advocate a social service group of courses involving both scientific and practical aspects of social service with sufficient emphasis upon the statistical side, with proper attention to the facts of economics and sociology as they now stand, but with tremendously greater emphasis upon the psychology of the instincts, the emotions and the will. I should like every practical social worker to be cognizant of language difficulties, if not in command of a speaking knowledge of some other language than his or her own. This problem deserves to be taken with great seriousness. I believe that the social service group of interests is at least as important as the natural science group and the literary or humanities group.

The field of social work may be considered in three main divisions, — work with the individual, community work and research. An example of the last is the Federal Children's Bureau, which employs a number of skilled investigators to collect social data for educational and legislative purposes. Community work includes all social movements that deal with people in groups or masses. Work with the individual, or social case work, as it is usually called, is the division of social work in which the biggest and most immediate demand will be created by the war, through the need of care for convalescent and disabled soldiers and of assistance to the dependent families of enlisted men.

Social case work has methods and principles which can be learned only by its practice. Its object is to restore socially disordered individuals as nearly as possible to normal life. This requires first a study of the individual, and next the organization of all factors in his life, both within himself and in his environment, to effect as complete an adjustment as is possible of that individual to society. This process is fundamentally the same whether we are dealing with a dependent child, a deserted wife, a delinquent girl, a tubercular patient, a psychopathic patient or a crippled soldier. In each case you are dealing not only with the original client, but also with his family, and in each family group you may find any one of the other types. The type of case you specialize in will predominate, but all types will appear. In the eight months' course of study and practice in psychiatric social work that we offer at the Psychopathic Hospital we believe that the student gets experience that prepares her for any form of social case work in addition to special knowledge of mental disease. All branches of social case work deal with psychopathic individuals, recognized or unrecognized as such. On the other hand, the patients of a psychopathic hospital are subject to the same social disorders that the clients of other agencies are. The elements of training should be the same for all case work. The great need is for workers disciplined in the methods and principles that have so far been developed. One valuable book, which appeared this year, is available on this subject.[1] No large body of experience has yet been systematized, and it is still common to hear that a mature able woman will be as successful as the trained worker. But any supervisor of case work will tell you that there is a distinct and important difference between the work of an inexperienced person of ability and an equally able person with training. The popular idea that case work was merely a matter of common sense and intuition has no doubt been responsible for a sentiment, just beginning to evaporate, that case work was a rather inferior part of social work. A social worker until recently was supposed to be "advanced" from case work to an executive or research position. It is now becoming recognized that the study of an individual out of adjustment with his environment and the organization of his life to effect normal adjustment may be a science fit to engage all the powers of mind and personality that a man or woman can bring to it.

Colleges and Special Schools that offer Courses in Social Work.

The New York School of Philanthropy, United Charities Building, 105 East 22d Street, New York City.
Chicago School of Civics and Philanthropy, 2559 South Michigan Avenue, Chicago, Ill.
Boston School of Social Work, 18 Somerset Street, Boston, Mass.
Pennsylvania School for Social Service, 425 South 15th Street, Philadelphia, Pa.
St. Louis School of Social Economy, 221 Locust Street, St. Louis, Mo.
Carola Woerishoffer Graduate Department of Social Economy and Social Research, Bryn Mawr College, Bryn Mawr, Pa.
Department of Social Work, Indiana University, Indianapolis, Ind.
School of Applied Social Sciences, Western Reserve University, Cleveland, Ohio.
Toronto Training School, University of Toronto, Ontario, Can.
Richmond School of Social Economy, Richmond, Va.
Margaret Morrison Carnegie School, Pittsburg, Pa.
New Orleans School for Social Workers, 1202 Annunciation Street, New Orleans, La.
Jewish School for Communal Work, 356 Second Avenue, New York City.
Social Service and Nurses' Training School of Georgia, 23 East Cain Street, Atlanta, Ga.

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THE FAMILY OF THE NEUROSYPHILITIC.*

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AND

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It may seem trite to call attention to the problem offered by the family of the syphilitic, as it is recognized by all that syphilis is a contagious disease and that in consequence it is frequently transmitted by the infected person to the spouse and to the children. In spite of this knowledge, however, the problem is largely neglected.

The complete story of syphilis and its ravages cannot be told in a limited space. Suffice it to say that the symptoms of syphilis may occur at any time after the infection, even though an individual live a half century, and that syphilis imitates in its symptoms almost every disease. Some of the most frequent and most serious of the syphilitic conditions are cardio-vascular disease, *i.e.*, *early* arteriosclerosis, apoplexy aneurysm, angina pectoris, etc., and diseases of the nervous system, such as general paresis, locomotor ataxia, cerebrospinal syphilis. The latter conditions, due to syphilitic involvement of the central nervous system, are what we include under the term neurosyphilis. General paresis and locomotor ataxia (tabes dorsalis) are always and without exception syphilitic manifestations. Just as without the tubercle bacillus there can be no tuberculosis, so without the spirocheta pallida (the germ of syphilis) there can be no paresis or tabes.

We cannot here consider the mental, physical and moral devastations, the economic and social wastage, the anguish and suffering caused by syphilis in those who have acquired it in adolescent or adult life after nature's defences have been broken down. Nor can we do more than merely enumerate a few of the terrible consequences passed on to the progeny of the infected. Sterility, abortions, miscarriages, stillbirths and early deaths are among the less terrible consequences of parental syphilis; these potential lives escape the suffering and handicaps of many who are born with congenital syphilis. Feeble-mindedness, deafness, blindness, paralysis, deficient development, marasmus, meningitis and skin diseases are among the early manifestations of congenital infection. Many congenital syphilitics are afflicted only with lessened vitality, anemia, delayed development, irritability, nervousness, neurotic manifestations and the like. Others are apparently healthy, well-endowed children, but during the pubescent and adolescent periods the presence of the disease first makes its appearance, frequently as interstitial keratitis, leading to partial or total blindness, or deafness; or there may appear the symptoms of syphilis of the central nervous system, known as juvenile paresis, juvenile tabes, etc., running through the whole gamut of the conditions produced by acquired syphilis. Many more conditions resulting from congenital syphilis might be added as well as the suspicion that many cases of neurasthenia, hysteria and dementia præcox may be of like cause; but enough has been said to indicate the ravages of the disease.

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* Read at the National Conference of Social Work, Pittsburg, 1917. Reprinted from Mental Hygiene, Vol. II, No. 1, January, 1918.

How is it possible that such conditions are allowed to continue almost without challenge? The reasons are several. The greatest of them is ignorance upon the subject, not only of the uneducated public, not only of the semi-informed social worker, but also a considerable degree of ignorance on the part of many physicians. This is not an arraignment of physicians; it is only a frank acknowledgment that our information is but slowly accumulating, and is still almost entirely in the hands of the specialist. To any one suffering from syphilis, let us boldly offer the advice that none should be discouraged if his physician disagrees with what is here stated; consult a specialist in syphilis and he will bear out these observations. We can hardly pause to recall the difficulty of lay education on this previously prohibited topic and the physician's hesitancy of betraying in any way the confidence of his syphilitic patient, even to protect another.

But there are other factors to be considered which depend on the very nature of syphilis. After the early symptoms, for a long period of time there may be no evidence to the patient that the disease is active, and hence he thinks he is safe. Even though warned by a physician he may disbelieve and marry; or the physician may believe him cured and give consent to marriage; or he may ask no advice; or the future spouse may know and not object to marriage on this trivial score; or the victim of the disease may be ignorant of the fact that he has or had acquired syphilis.

At any rate, as we well know, marriage is entered into by many syphilitics, and then arises the problem of the family of the syphilitic. This problem as previously stated is well recognized by all engaged in the study of syphilis. The family of the syphilitic, including spouse and children, has been exposed to syphilis, the spouse exposed to the so-called acquired form, the children to the congenital form. Not all exposed to syphilis acquire the disease — far from it, for at least 75 per cent. of all prostitutes have syphilis, and if it were universally infective it would be almost universally widespread. There are certain laws that give us some insight into the infectivity of syphilis; for instance, it becomes less contagious as time goes on, and open lesions are usually necessary for its transmission. In the congenital form the ravages grow less as the time from the original parental infection increases. These laws can be found in textbooks on syphilis, both medical and for the layman.[1] But these laws are based on statistics, and while they apply to a group of cases, any particular case under consideration may prove an exception. In other words, there are many exceptions and much that we do not understand, so that every case presents a special problem.

Syphilis in both its acquired and congenital forms is not always easy to recognize. A characteristic of syphilis is that there are long periods of quiescence or latency lasting months and years, during which there are no symptoms that bother the patient or appear on superficial examination. Then five, ten, fifteen, twenty or even forty years of latency is broken by an exacerbation or a cropping out of symptoms, — symptoms perhaps easily recognized as syphilitic, but recognized often too late for treatment. In other words, latency may only mean the absence of manifest symptoms, while the disease is actively at work in destroying the organism. This period of latency is to be found in nearly every case in which the most serious results occur. Many individuals are not aware that they have acquired syphilis until these late manifestations appear. We have often had patients relate how lucky they had been in avoiding syphilis all their lives, when as a matter of fact they were victims of the disease.

It is during these years of apparent latency that much can be done by treatment to prevent the late symptoms of syphilis, and it is for this reason that we desire to discover cases that are existent but unrecognized. On the whole, the earlier treatment is instituted for syphilis the better the results. But it is not entirely easy to make a diagnosis of syphilis. In the last few years we have been greatly helped by the Wasserman test, but this is not a sufficient and final criterion. There is still much difficulty and much to be learned. But we do know that the most likely field for the discovery of syphilis is the family of the syphilitic patient. Therefore the modern, well-equipped and well-managed syphilis clinic makes the study of the families of the patients of the clinic an integral part of its service.

The examination, and treatment when indicated, of the family of the syphilitic becomes one of the greatest services such clinic offers the patient, his family, the community and the nation. As has been said, such plan is being followed to-day in the majority of syphilis clinics, but unfortunately only a very insignificant percentage of cases of syphilis ever reach the syphilis clinic. Many cases found in other parts of a hospital are not sent to the syphilis room nor thoroughly considered. Many cases are not recognized; many are treated by private physicians; many are in orphanages or with child-helping agencies. The beginning has, however, been made; the problem has been outlined; and many have become initiated into the circle of workers and so the work will increase.

But the problem we have set ourselves is to speak of the family of the neurosyphilitic. After what has gone before it can be quickly and briefly stated. The problem of the neurosyphilitic's family is the problem of the syphilitic's family. Then why say more? Is it merely an excuse for emphasizing the need of family treatment? That is only partly the reason. It is because it is rarely realized that the family of the neurosyphilitic is the family of the syphilitic. And so, purposely, we reiterate — the family (the spouse and children) of the general paretic is the family of a syphilitic, and the family of the tabetic is the family of a syphilitic. And thus these families become the heirs of all the scourges of the syphilitics, as briefly indicated previously.

To understand why it is necessary even to consider what ought to be a commonplace fact, axiomatic, accepted and acted upon, we must briefly consider the development of our knowledge concerning general paresis, tabes and other forms of syphilis of the nervous system. It must be remembered that the symptoms of the syndromes making the classification of general paresis and tabes occur years after the infection, and follow a long period of quiescence or latency in which the patient is free from gross symptoms. It was not until very recently that these conditions were recognized as syphilitic. At first a few students of the subject called attention to the frequency with which paretics and tabetics gave a history of syphilis. The suggestion of Moebius, little more than a generation ago, that paresis and tabes were syphilitic conditions in 100 per cent. of the cases, met with very great skepticism, but as our information increased and as new tests were discovered this hypothesis became more and more plausible. It seemed, however, too simple an explanation, too good to be true, as it were, and a loophole had to be found. This was furnished by Fournier, the great French syphilographer, who taught that paresis and tabes were the result of syphilis, but were not manifestations of active syphilis, occurring only where syphilis had previously been. He coined the term "parasyphilis" to cover this conception, and this term is still with us, though we have learned that its original conception was based on a false premise. This idea concerning paresis and tabes as parasyphilitic did much harm in diverting attention from the problems of syphilis, and its damaging influence is still only partially dispelled. It was not until 1913 that the final link in the chain of evidence that parasyphilis is active syphilis was forged by the discovery of spirochetes in the brain and cord of the paretic and tabetic by Noguchi and Moore.

Harmful as was the teaching of Fournier, it had the effect of stimulating men to disprove it, and led to the examination of the families of paretic and tabetic patients for further evidences of syphilis. With the discovery of a serum test for syphilis in 1906 by Wassermann, Neisser and Bruck, a new aid and a new impetus was given, and since then studies have been made by men in all countries, among whom may be mentioned Nonne, Régis, Haskell and Hyde. The findings differ only in percentages, varying with standards and methods; all agree that the devastation is great, the menace serious. Yet, despite the evidence on all sides, we occasionally hear, from those who have not themselves thoroughly studied the matter, statements that the children of paretics and tabetics who do not die early grow up into exceptionally fine specimens of humanity. It is because of such statements that we have felt justified in doing more than emphasizing what seems to us so obvious, namely, that spouses and children must be carefully studied for manifestations of syphilis. We therefore give herewith an analysis of our work in the families of syphilitic patients suffering from nervous system involvement, and compare them with those where the central nervous system is not involved.

It is a routine at the Boston Psychopathic Hospital to examine whenever possible the families of all our syphilitic patients. To do this it is necessary to get the spouse and children of the patient to report to our out-patient department. To obtain this end the aid of the social service department is invaluable, and without such aid it would be almost impossible to do the work adequately or efficiently. With such aid the majority of the individuals desired readily co-operate for examination and treatment.

We have thus far examined the families of 247 syphilitics, of which 160 families were of syphilitics suffering from general paresis, 72 of syphilitics without definite central nervous system involvement, and 15 with the diagnosis of tabes and cerebrospinal syphilis.

In the 160 families in which one parent was a paretic, 226 individuals were examined. If we consider the Wassermann reaction alone we find that it was positive in 23 per cent. of the group, or in 52 individuals. Of these 52 positive Wassermann cases, 33 were spouses and 19 were children suffering from congenital syphilis. It should be mentioned here that the Wassermann reaction is only a part of the examination. Many patients with syphilis give a negative Wassermann reaction. However, it is given preference here as having less error on account of the individual equation in examination, and because it is better standardized.

From the 72 families in which one parent had syphilis but no evidence of central nervous system involvement, 91 members were examined. In this group 35 per cent. gave a positive reaction, — more than 1 in every 3. Of these positive reactions 13 were found in the mate of the patient and 18 were congenitally syphilitic children.

The group of tabetic and cerebrospinal syphilis families is too small to merit consideration here, further than to say that the figures of syphilitic involvement ran slightly higher than in the other larger groups.

These Wassermann findings rate much higher than in the population at large, where the percentage of positive reactions is given by various investigators as varying from 5 to 15 per cent. In the

Psychopathic Hospital the average varies each year between 12 and 14 per cent. In the groups under consideration it ranges from 23 to 35 per cent.

One wishes to know, however, in what proportion of the families this positive finding occurs. It might be that a large number of syphilitics occurred in a few families, thus producing the relatively high percentage. But that is not the case. In the whole group of 247 families a positive Wassermann reaction was found in 68 families, or 27 per cent. In the 160 families of paretics it occurred in 45 families, or 28 per cent., while in the 72 families of non-nervous system syphilis it occurred in 19 families, or 26 per cent.

It should be borne in mind that the Wassermann findings do not represent the total of syphilitic involvement. In many syphilitics a negative blood Wassermann is obtained. Much of the syphilitic damage is represented by abortions, miscarriages, stillbirths and early deaths. No greater cause of race suicide can be imagined than syphilis. For example, of the group of 247 families 84, or 34 per cent., were sterile — had no children; of the 160 families of paretics 53, or 33 per cent., had no children. In the population at large the least productive group as to children is supposed to be the college-trained, but we find among the Harvard and Yale graduates only from 19 to 23 per cent. of infertile marriages in contrast to the 33 per cent. among paretics. It was found, further, that 20 per cent. of the families had abortions, miscarriages and stillbirths, while dead children occurred also in 20 per cent.

As a result we find the average of living children per family to be 1.3, a figure very much lower than necessary to keep the population stable. The figures are about the same for families in which the original patient had or had not nervous system involvement. The birth rate for the 247 families averages 1.7 children. From the United States census report it is found that the average birth rate in our vicinity is 4.4 children per family. But this difference betwen 1.7 and 4.4 does not tell the whole story. Were it not for syphilis the 4.4 figure would be higher, for that average includes the syphilitics. But of the 1.3 living children per family, many are afflicted with syphilis and will have a shortened life and a lessened efficiency.

To sum up, incompletely, what syphilis has meant to these 247 families, it will suffice to state that only 69, or 28 per cent., showed no defect as to children, 30 per cent. in the paretic and

27 per cent. in the other group. Only 61 families, or 25 per cent., showed neither defect as to children nor Wassermann reaction. These figures, it seems to us, show unequivocally that the incidence of syphilis is tremendously high in the families of the syphilitic, and that it is as high in the families of the paretics and tabetics as in those whose syphilis has not invaded the nervous system.

A few illustrations may be given as concrete instances.

Family 1. — A woman came to our attention suffering from general paresis. On examination of the other members of the family it was found that the husband showed the early signs of syphilis of the nervous system. The oldest son was brought to us with juvenile paresis well marked, and died four months after first being seen by us. The second son died a month later of ruptured aneurysm, at the age of 20, certainly due to syphilis. The third son had the stature of an achondroplasia. The fourth was a mis-shapen cripple with caries of the spine, while the fifth son although in good health, showed stigmata of congenital syphilis. There were three stillbirths.

Family 2. — A boy of fourteen, an only child, was brought to the hospital because he was backward mentally. He was found to have juvenile paresis. The father on examination proved to be in the early stages of locomotor ataxia. The mother was quite deaf, due to syphilitic involvement of the eighth nerve.

Family 3. — A man with general paresis had a wife and two apparently healthy children, all three having positive Wassermann reactions.

Family 4. — The husband had paresis; the wife showed no evidence of the disease. The older child, aged six, was feeble-minded and had a positive Wassermann reaction; a younger sister, aged four, was apparently bright and healthy, but also had a positive Wassermann reaction.

Family 5. — A woman came to us with her two daughters, aged eleven and nine, asking to be examined. Her husband had recently died of general paresis. She had heard that this was a syphilitic disease. From Brieux's "Damaged Goods," she had learned of congenital syphilis and hence her request for examination. She proved to have a positive Wassermann reaction. The older girl was anemic, underdeveloped, nervous, irritable and bore the unmistakable signs of congenital syphilis. She had a positive Wassermann reaction. The younger child was apparently well endowed mentally and physically, but also had a positive Wassermann reaction.

But why should we make these examinations that show such unpleasant findings? It is in order to help these sufferers from syphilis, who for the most part are innocent and unsuspecting victims. The characteristic of syphilis, mentioned above, to remain dormant or apparently inactive for a long period gives us the opportunity, if we grasp it, to apply therapy before the ravages have, in some instances, gone very far. Hence our desire to make the diagnoses early. Then therapy is of avail. In Family 1, it was obviously impossible to do aught for the first or second son, but probably much could be done for the fifth to keep him from going the path of his older brothers.

In Family 3 we have a striking example of the value of such examinations. Here is a mother and two children who, while apparently healthy, are suffering from syphilis. Treatment applied now may well prevent any future symptoms. The aim of medicine to-day is first, to prevent disease, and where that is not done, to discover it as early as possible, that treatment may have its greatest chance for success. By diagnosis, made possible by family examination, something can be done to prevent the spread of the disease; much can be done to prevent symptoms, and hope can be offered for healthy children by treatment applied to the parents before the birth of children, and often to the children themselves when no great damage has yet been done by the disease.

It is not our province to point out the methods of diagnosis or treatment. This, in every instance, is highly specialized work for the physician, but it may be well to insist that results of therapy in syphilis, and especially in late and congenital syphilis, can be obtained only after years of treatment, and that the patients must be under medical care probably for life.

To handle the problem properly education is essential, — education of the physician, the social worker and the layman. The value of education of the layman is shown by the illustration (Family 5) in which the mother, through her reading of "Damaged Goods," sought aid, when such aid had been withheld by physicians who had not done their duty in diagnosing the children as syphilitic.

As an argument against family examinations in syphilitics, it is sometimes said that there is grave danger of breaking up the family if it is learned that one member has syphilis. This is a very weak argument against examination, we should say, if by such examination steps can be taken to eradicate syphilis from the family. But the argument is *entirely* fallacious. In three years' experience with the families of syphilitics, and in dealing with more than 250 families, we have not broken up a single family, nor, we believe, brought undue unhappiness into any. We shall be able to begin to attack the problem of syphilis only when the knowledge of syphilis and its manifestations and ravages is widespread. To those who fear to tell a man or woman that general paresis and locomotor ataxia are syphilitic diseases we would give warning that before long this will be as common knowledge as that consumption means tuberculosis. And to those who have withheld this knowledge and allowed syphilis unhindered to reap its harvest, not thanks but rather recriminations will come.

In closing we would repeat that the families of syphilitics show syphilis in a very high percentage; that the families of paretics and tabetics are the families of syphilitics; and that the families of all syphilitics should be thoroughly examined that treatment may be instituted where indicated.

REFERENCE.

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A WIDER FIELD OF ACTIVITY FOR THE AMERICAN MEDICO-PSYCHOLOGICAL ASSOCIATION.

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The American Medico-Psychological Association has a dignified history of nearly seventy-five years of continuous activities. Founded in 1844, it has consistently maintained standards which have made it one of the most prominent of the medical organizations of the country. It was conducted for nearly fifty years as the official organization of the superintendents of the American institutions for the insane, and only adopted its present designation after the reorganization in 1892, when its membership became more general.

It now includes as active members physicians "especially interested in the treatment of insanity," or who "by their professional work or public writings have shown a special interest in the care and welfare of the insane." Laymen who "have distinguished themselves by their attainments in branches of science connected with insanity, or who have rendered signal service in philanthropic efforts to promote the interests of the insane," are eligible to honorary membership. The association has assumed a position of prominence among the scientific organizations of the country, and includes in its membership practically all of the prominent American psychiatrists of the day.

It has, however, unnecessarily and unwisely relinquished its official representation of the various State bodies responsible for the management of the institutions, public and private, for the insane. In so doing it has failed to take advantage of the opportunity of encouraging and assisting in the maintenance of high standards of care throughout the country, of influencing legislation conducive to the welfare of the insane, and of guiding the destinies of the institutions as it should.

The association should be the most powerful factor in the country in obtaining State recognition of the needs of the insane, in fostering legislation which will prevent political interference with the hospitals, and in bringing the standards of care up to the high plane represented by ideals kept constantly before the public by this organization since its foundation in 1844. If this is to be done we must include in our membership the men who control the policies of the institutions in the various States.

For this purpose I would suggest for your consideration, if necessary, an amendment to the Constitution which would render eligible, *ex officio*, as active members all duly elected or appointed managers or trustees of State hospitals for the insane, and all members of commissions and boards of control having administrative or financial responsibility for State institutions for the insane, such eligibility to continue while actually in office. They should, of course, be required to pay annual dues and be subject to all the requirements of the Constitution applying to active members. If the association is not to be an official organization representing the institutions for the insane, and it has not been since 1892, it should at least include in its membership the managing and governing bodies vested with the administrative control of the hospitals in the various States and provinces. This subject might very properly be referred for further consideration to a committee to be appointed by the president.

The association should, in my opinion, be something more than a purely scientific body, meeting annually for the presentation and discussion of papers relating to psychiatry. It should officially exercise the functions which the professional qualifications and official position of its active members would fully warrant. No other one thing would go so far towards insuring the humane and proper care of the insane in this country.

The report of the committee on statistics at this session calls attention to another phase of activity of the association which has been under consideration for many years. At the twenty-third annual meeting, in 1869, Dr. Nichols called the attention of the association to a system of statistics adopted at the International Congress of Alienists held in Paris in 1867. A series of statistical tables, twenty-one in number, were prepared by a committee and used unofficially for several years, although for some reason never formally adopted. Another committee reported on this same subject in 1896, unfortunately without any definite action.

At the present time we are without any statistics on the insane of the country which can be correlated and made a subject of scientific study and investigation. With a wealth of material available which would go far towards solving many important problems relating to psychiatry, we find the various States, and more often individual institutions in the same State, working along entirely dissimilar lines.

Our textbooks are filled with uncorroborated assertions relative to the frequency of certain forms of insanity. These are often based on the personal experience of the author or on statistical data available from his own hospital. The official publications of a few isolated States give such information in an intelligent form, but any one who wishes to compare conditions existing in one community with those in another, or make comparative studies of large numbers of cases, almost invariably finds the necessary information unavailable.

Accurate statistical data showing the etiology of insanity are published in but few places, and the discussions of this subject are based more on opinion than on the study of a sufficient number of cases to warrant any conclusions. The same absence of authentic information renders it impossible to obtain any accurate knowledge regarding the recovery rate of the different psychoses. Dementia præcox is conspicuously absent from many of the hospital reports which include primary dementia, chronic delusional insanity, terminal dementia and other conditions which may or may not belong in the dementia præcox group.

When statistical reports are made they usually contain information which is of itself very largely useless relating to the civil condition, color, sex, age, residence by counties, financial status, etc. Unless the psychoses are shown in these tables they are of no value whatever. In any event, it is impossible at the present time to obtain statistics regarding any one phase of insanity which is of any importance from more than a very few States where the insane population is unusually large.

Accurate statistical reports made along uniform lines would enable us to reach important conclusions regarding the frequency of occurrence of the various psychoses, the probability of recovery, the intercurrent affections causing death, etc.

A uniform basis for financial reports made annually to the association would be of incalculable benefit. This would not conflict in any way with returns made to local fiscal officers. Our attention is often called to the apparent fact that the per capita cost of maintaining the insane in certain States is unreasonably high, whereas they are cared for at a much lower rate in other communities. This has frequently been made the subject of legislative investigation, usually with highly misleading conclusions owing to the fact that we have no uniform way of computing costs. Some institutions in reporting the cost of maintenance deduct from the gross expenditures the receipts from all sources, including the care of reimbursing patients and the value of all farm and manufactured products. Others very properly do not. Some include only the ordinary repairs and replacements, and others the cost of new and additional equipment, extraordinary repairs and even additions to buildings. The result is that, if the statements published in annual reports are taken at their face value, the same methods of administration that would appear penurious in one institution would be interpreted as gross and almost criminal extravagance in another.

The reports of the State Hospital Commission in New York have been enlightening as to the burden of maintaining aliens in our public institutions. It has been shown that there are now about 9,000 patients in the hospitals of that State who are not residents of New York or citizens of the country. The actual maintenance of these aliens is costing the State approximately $2,000,000 per year. This is undoubtedly a condition which exists in practically the same relative proportions in the other States. The facts which are necessary for an intelligent discussion of this important question are, however, lacking because this subject has not been generally included in statistical reports.

The national and various State mental hygiene societies have been endeavoring to educate the public regarding the importance of the relation existing between insanity, syphilis and alcohol. Unfortunately, statistics on these subjects cannot be obtained in many of our larger communities.

In view of the objects and purposes of the American Medico-Psychological Association it would appear to be the obvious duty of this organization to endeavor to remedy this unsatisfactory condition of affairs. The report of the committee on statistics has pointed out the way in which this can be accomplished. The approval by the association of a scheme of uniform statistical reports of the insane will unquestionably insure the success of such a movement, and a concerted effort on the part of our members is quite sufficient to bring about the adoption of these statistical forms in the various States and insure their general use.

I would strongly urge upon the association the advisability of establishing a bureau of statistics for the purpose of compiling and publishing annual reports on the insane in all the States and provinces as far as obtainable. Such a bureau could probably be maintained at a comparatively small expense in cooperation with the National Committee on Mental Hygiene.

The success of "The Institutional Care of the Insane in the United States and Canada" shows conclusively that undertakings of this magnitude are entirely practicable if the association will assume the function which properly devolves upon it of correlating the work done by the various States. It would appear to be practically certain that the Federal authorities can be induced to arrange for a census of the insane based on our official classification, which will, for the first time, furnish us with intelligent information on this important subject. If the association accomplishes nothing else this would constitute one of its most important contributions to medical progress.

ON THE FOCALITY OF MICROSCOPIC BRAIN LESIONS FOUND IN DEMENTIA PRÆCOX.

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Abstract.

The task of microlocalization.
Work of the topographers.
Problems of a pure functionalism.
Anomalies in dementia præcox brains.
Crucial importance of histology of normal-looking brains in dementia præcox.
Dissociation of parenchymatous and interstitial lesions in certain cases.
The stratigraphical analysis of the cerebral cortex, supracortex and infracortex.
Many effects of mental dissociation (schizophrenia), probably correlated with supracortical disease.
Correlations of hallucinosis and catatonia with infracortical disease.
Topographical analysis; *à priori* probabilities.
Tendency to lobar and lobular focality of lesions.
Delusions: frontal; catatonic symptoms; parietal; auditory hallucinosis; temporal.
Exceptional delusions of a fantastic nature probably parietal in origin.
Analysis of four cases.
Summary.

The present attitude of investigators of the mind and its diseases has to pursue the study of the nervous system along lines roughly summed up in the term microlocalization. But this task must not be conceived narrowly as a task of the histoneurologist or the histoneuropathologist. Microlocalizations in the nervous system and especially in the cerebral cortex are threads running to the broadest issues of physiology, embryology and psychology. Yet no one feels that psychologists, embryologists, physiologists, anatomists, or even pathologists in the narrower sense of the term, will solve the problems of microlocalization or even demarcate its problems for solution. When the idea of Flourens, that the brain parts were mutually exchangeable in function like so many liver parts, was replaced with the ideas of topographical differentiation, which we attach to the work of Hughlings-Jackson and of Hitzig, it was as if the naked eye of observation was replaced with a lens of considerable power. The situation now is that this lens of great power, provided by the work of the embryologists, the physiologists and the clinical anatomists, must be replaced with a lens of still greater power, one that will permit us logically to face problems of microlocalization thus attaching to physiological complications within the single gyrus. It has become a commonplace that the brain is a congeries of organs. It is not so well understood that each gyrus is a wonderland of structures and functions in which rich results are to be expected from hard but fascinating research.

As we look over the history of these developments we of course find that Bevan Lewis had, in advance of his time, laid down the idea of cell differentiation within the cerebral cortex, clearly distinguishing the large cells of the motor cortex, for example. In much of the work of Meynert and Flechsig the idea of microlocalization can be discerned. However, even in the embryological considerations of Flechsig the notion of the gyrus as little more than a functional unit is the prevailing idea. Decades were devoted to the isolation of one neurone after another by ingenious technical methods, and to developing the conception of the nervous system as a mass of embryologically and functionally separate units, expressed, for example, in the neurone hypothesis of 1891. Listeners to the lectures of Nissl became familiar with his ideas of the structural and functional differentiation of the various cortical parts.

The idea of microlocalization scarcely flourished to the full in the last decade of the nineteenth century, however, since workers were engaged in assimilating the principles of Weigert concerning neuroglia and in trying to find qualitatively the same sort of lesions in the nervous system that they were finding in other organs. In fact, this search was neatly culminated in the establishment in the years 1899–1904 of general paresis as a definite example of chronic inflammation of the nervous system. The pathologist was here trying to find and succeeded in finding certain lesions scattered in different parts of the nervous system, and lesions of qualitatively the same sort throughout. Nor in the rush of new ideas was the material in general paresis used to the utmost in throwing such light as it might on the problem of microlocalization. Thus, had the world been seriously trying to find out the nature and genesis of mental phenomena and symptoms, it would surely have executed elaborate studies in the distribution of the characteristic paretic exudate, with the idea of correlating these local variations in exudate with processes and symptoms. The issue of such an investigation could only have been positive or negative. As it stands, the situation is doubtful. In fact, it may be confidently doubted whether the paretic exudate in paresis is not itself responsible for many of the characteristic symptoms in paresis, for example; grandiosity, amnesia or even dementia, which are not found to correspond even roughly with the amounts of exudate discovered in the brains. So far as paresis is concerned, very possibly it was more important to determine it to be syphilitic and to embrace eagerly the opportunities afforded of a Wassermann reaction than to pursue seemingly recondite studies in microlocalization. Meantime the topographical anatomists were laying down the results which the pathologist could at last employ without signal loss of time.

When the interesting and fundamental work of Hammarberg appeared on the histology of feeble-mindedness in 1895 it was plain that this Swedish investigator had had to spend more than half his time, not on the histopathology of feeble-mindedness, but upon normal histology. The situation is now greatly improved with the work of Elliot Smith, who showed that even the naked eye will, under proper conditions, detect striking differences between the gyri of different parts of the brain on section, and especially with the work of Campbell who, after an extensive study of the histology of the different gyri, has offered a valuable atlas with a store of functional comments and speculations.

Even before Campbell, Bolton had laid down exact lines concerning the histology of different parts of the occipital region, and had contributed the idea of visuo-sensory and visuo-psychic regions therein. Similar work has been done by Brodmann in Germany, who, however, feels that functional speculations are hardly in point at the present time. Visitors in the private laboratory of Vogt in Berlin some years since must have been impressed with the tremendous elaboration of structures which could be demonstrated in the different parts of the brain, even by so simple a technical method as the method of Weigert for myelin sheaths. For decades, also, the world has been watching the development of the ideas of Ramon y Cajal, whose labors stand not second to those of Bolton, Campbell, Brodmann and the rest of the topographers.

In fact, the research situation is theoretically so promising in view of the progress along all these lines by workers in different countries that it is a little surprising that so little neuropathological application has been made of the results as attained. There seems to be a curious lack of tempo in neuropathological research. Bevan Lewis' ideas of cortex differentiation came, it would seem, too early to affect the main current of investigation. The work of Donaldson in 1889–90 upon the brain of Laura. Bridgman has not been followed up by any considerable series of similar investigations, and the Laura Bridgman work apparently led its author to deeper considerations of growth and development rather than to progress along the line of neuropathology.

Again, throughout the nineteenth century, there prevailed the Wundtian psychology. The reader of Wundt's analysis of aphasia, for example, is convinced that, despite the profound distinctions made, the philosopher was no longer a physiologist, and set himself the task of anti-localization in the spirit of a system-maker rather than that of an independent seeker after facts and independent interpretations. The idea of the unity of the mind, a very fine-grained and closely interwoven unity, has prevailed in many quarters, perhaps in all quarters, dominating research. To these psychic unitarians it would have seemed almost a pity if any one could show, for example, an identity of speech processes with processes of the left inferior frontal gyrus. To the conservative wing of the psychic unitarians was added the force of those strictly scientific persons who feel that a fact is a fact and a speculation is necessarily a bad speculation. Thus, for example, if the simpler ideas of Broca concerning speech localization ought to be modified by the modern concepts of Marie, then this is scored as a victory by those holding to the adamantine unity of the mind on the one hand, and by those who are skeptical of all scientific results in the psychic field of whatever sort.

Added to this situation was the Freudian tendency. Disregarding the truth or falsity of the Freudian contentions, it is clear that his followers are engaged much more in finding satisfactory categories of a logical nature in which to place globar tendencies shown by their patients than they are in conceiving the brain functional lines along which an hysterical dissociation might proceed, a repression be mechanized or a censor be enthroned.

Inasmuch as Descartes made a laughable error in assigning the seat of the soul to the pineal body, it is best for the modern to "play safe" by failing to notice both the pineal body and the brain itself, and to solve the problem either by denying that it exists or by failing to consider it at all. One of these modern worthies assured me some years ago that there was no doubt a correlation between mental processes and brain processes, and that theoretically the "needle" of demonstrable lesions could be found in the "haystack" of neuronic brain systems, but that for his part he regarded that problem as theoretically solved, and desired to get on with something genuine and practical.

Accordingly, the academic psychologists, if we may so term the Wundtians, and the Freudians, have alike little interest in the matter of microlocalization, that is, in the correlation of mental with brain processes, either normal or pathological.

It is therefore almost with a feeling of being alone in the wilderness that one endeavors to work in the direction of neuropathological correlation;. either there is no problem or it is theoretically settled beforehand. A psychiatrist of light and leading asked me one day what, after all, I was about in working along structural neuropathological lines. Anybody with a logical turn of mind and any capacity for observation ought rather to be busily applying the modern categories of psychopathology to clinical psychiatric and neurological material.

If such doubt of the fundamental value of this kind of work could prevail in the mind of a leading psychiatrist, I felt that I could do no better than bring these general ideas before the Association of American Physicians.

It is as possibly throwing light on the problems of microlocalization that the material in dementia præcox struck me as of value. To be sure, the world was full of a rather empty discussion as to whether dementia præcox was an organic or structural disease. Any one who professes to find brain changes in dementia præcox was perforce regarded as a controversialist, engaged in demolishing the ideas of some workers who wanted to show that dementia præcox was a disease of maladaptation, or a disease with so-called Freudian mechanisms. Let me say, therefore, that to embark on studies of the brain in dementia præcox and to find brain changes therein is not at all to deny that dementia præcox is an instance of maladaptation of the individual to his environment, and not at all to deny the possible importance of Freudian mechanisms in the disease.

In 1906 I began to study the brain problems of dementia præcox, and collected first fruits of that study at the Medical Congress in 1910 and 1913.[3] I then showed that the brains of dementia præcox subjects were extremely likely to show evidences of anomaly; that even when there was no anomaly there were very likely to be microscopic changes. To be specific, I found 23 out of 25 carefully studied brains to be significantly anomalous. I do not mean that the anomalies shown were visible the whole length of the demonstration hall as they are apt to be in brains of the feeble-minded, but I mean that the asymmetries and irregularities were of a demonstrable and photographable nature. Friendly critics offered the suggestion that similar anomalies were to be found in brains of normal persons. Unluckily the brains of normal persons, in large numbers, are not available so far as I know in any laboratory in the world, at least in such a state as to permit proper photographic analysis. However, I think I am able to answer these critics by showing that the brains of subjects of the so-called manic-depressive psychosis show such anomalies in very few instances. Using certain criteria, I found four-fifths of my dementia præcox brains to show such anomalies, and but one-fifth of the manic-depressive brains were similarly affected.[5]

But it is one thing for a brain to be anomalous and another for it to be functionally disordered. The underlying hypothesis here is that the anomalous regions of the brain are in some sense weak places therein, such that disease of a toxic or metabolic nature, *e.g.*, at puberty, may unfavorably affect the anomalous and poorly constructed region. These considerations I presented with some histological confirmation at the 1914 meeting of the association in the paper "On the Direction of Research as to the Analysis of Cortical Stigmata and Focal Lesions in Certain Psychoses."[6] Further reflection on this finding led me to the considerations about tissue decomplication developed at the 1915 meeting.

But it was clear that the few cases of normal-looking brains in dementia præcox formed the crux of the situation, since the critics of material from anomalous brains might properly charge the investigator with inability to cull out the acquired from the inborn lesions. I therefore determined to study with sufficient intensiveness the brains of cases of dementia præcox that showed no anomalies or scleroses. In connection with this study five brains were investigated, four of them looking in the general direction of dementia præcox and one of them in that of manic-depressive psychosis. These five brains were the residuum of a series of 120 coming from psychoses of all sorts.[4]

Before I could bring the topographical problem into clear relief I found that in the analysis of these five brains the problem of parenchymatous *versus* interstitial lesions stood out. At the 1916 meeting I presented the conception that in the nervous system there should be a dissociation of lesions somewhat similar to that in the kidney.[7] To be sure, the parenchymatous and interstitial lesions of the kidney are, as is well known, often if not almost always commingled. But the fact that the lesions in these two types of renal tissue are so commingled in a given case militates not at all against the idea that there are, roughly speaking, two kinds of nephritis, — parenchymatous and interstitial. The same theoretical situation holds in the nervous system. I found that parenchymatous and interstitial lesions could be dissociated and combined in the nervous system much as similar lesions could be dissociated and combined in the kidney. To be sure, the parenchyma of the nervous system was a parenchyma composed of neurones and their adnexa, and the interstitial tissue of the nervous system was complicated by the fact that a great deal of it is neuroglia tissue, embryologically different from the interstitial tissue of the kidney. Nevertheless, it is well known that many of the properties of neuroglia of ectodermic origin are similar to the properties of mesodermic interstitial tissue. The alterations of the mesodermic interstitial tissue, also existent in the nervous system, are of far less consequence than those of the neuroglia tissue. The one case of manic-depressive psychosis, intensively examined, failed to show convincing degrees of parenchymatous lesion, but the dementia præcox case had well-marked parenchymatous disorder, that is, varying degrees of cell loss, to which the interstitial reaction, that is, gliosis, was found to be not at all proportionate. The result of this study seemed to be that parenchymatous disorder, *e.g.*, cell loss and interstitial disorder, *e.g.*, gliosis, must be investigated separately if we are to make headway in this group of cases. Thus it would be decidedly unwise to argue from the data of neuroglia preparations that the gliosis of such and such an area was an indicator of parenchymatous loss. To be sure, the gliosis is probably an indicator of something of general or local significance in the brain. But no facile application of the principle suggested by Weigert's neuroglia work could safely be made. Even if gliosis, as a rule, signifies some degree of wear and tear on the part of the parenchyma of the part affected, yet there was no good evidence that the gliosis was at all proportionate to the parenchymatous wear and tear.

Having thus shown that the brain conditions roughly corresponded to, *e.g.*, renal conditions, and that the histopathological analysis of injured brain tissues must look in both these directions in search of successful correlations, I proceeded to a stratigraphical analysis of the finer cortex changes in these brains, presenting results at a meeting of the American Neurological Association.[8] I tried to study separately the nerve cell losses on the one hand, and the gliosis (including satellitosis) on the other hand, and then to learn whether the character of these changes in the upper and lower layers of the cortex was of any functional significance. For convenience, I wish to term the upper layers of the cortex supracortical and the lower layers infracortical. In fact, it might be well at times to speak of the supracortex and the infracortex. The comparative anatomists seem to have demonstrated the importance of some such distinction, and the literature has for some time contained references to the so-called suprastellate (supragranular) and infrastellate (infragranular) layers of the cortex. Omitting controversial details, it appears that almost all workers are committed to the idea that the majority of the supracortical structures are of more recent evolution than the majority of the infracortical structures. As many of the mental functions in man are of recent evolutionary origin, and as all signs point to the cerebral cortex as somehow engaged in these processes, it seems natural to assume that the supracortical layers, or what we may term the supracortex, is the basis of many of these higher psychic functions. On the same plausibility we may argue that the infracortical layers, or infracortex, is the basis of such mental or quasi-mental functions as are found in those animals possessing an infracortex and not yet a supracortex.

All these interpretations are independent of any topographical conceptions, and for the purposes of generalization one neglects for the moment the important distinction of the archipallium and neopallium, developed by Elliot Smith.

Taking the whole cortex, by and large, modern work seems to me to invest the supracortex with higher psychic functions than the infracortex.

Now in a disease like dementia præcox as psychologically formulated by Bleuler we find the main feature to lodge in what Bleuler terms schizophrenia. This most valuable term expresses what one might call "split-mindedness," or mental dissociation. Much of modern psychology, whatever may be said to the contrary, is founded on the work of the associationist school of, *e.g.*, John Stuart Mill. We should hardly get on without some of Mill's so-called mental chemistry. Well, for better or worse, associative processes must have their obverse in dissociative processes, and the measure of mental dissociation is the degree of schizophrenia in Bleuler's sense. Kræpelin himself who, on purely clinical grounds of a combination of certain symptoms in certain ways, laid down the conception of dementia præcox, has so far admitted the validity of Bleuler's conception of schizophrenia as to use the term schizophrenic very frequently in his description of patients. In one of the most exquisite examples of schizophrenia, viz., the finely divided cleavage of speech known as *Wortwirrtheit,* Kræpelin has constructed the term "schizophasia" on the analogue of schizophrenia.

We mean, then, by schizophrenia a process of mental dissociation of a psycholitic or ideolitic process. The cleavage may lie between the intellect and the emotions, so that the emotions do not at all fit the ideas entertained by the patient; or the cleavage may lie between the emotions and the will, and a bizarre conduct may fit a certain quasi-normal state of feeling. But the cleavage is not necessarily so elective and total. The cleavage may be within some particular train of thought. Here is schizophrenia in its best display. Instead of the regular train of thought we may have a train of thought like a telegraph despatch, or even a train of thought suggesting in its obviously inappropriate sequence a cypher or a set of types reduced to pi. But absolutely pied thinking is not the rule, and the so-called word "salads" are often but passing phases in the development of a dementia præcox course. Here is not the place to expound or to describe schizophrenia. Inasmuch, however, as many of the schizophrenic effects that strike the observer as so bizarre in dementia præcox thinking are probably correlated with the operations of the outer layers, that is, of the supracortex itself, you have to inquire whether cases of this order show lesions preferably in the supracortex. Of my four cases of dementia præcox all showed lesions in the supracortex in the nature of cell loss and of more or less disproportionate gliosis, except one case. This case was one approximating the so-called paranoia, a disease perhaps allied to dementia præcox, but often running years or throughout life without any of the bizarre psycholitic, schizophrenic, thought-splitting phenomena here under discussion for paranoiacs of this rare description (there may be in a dozen institutions in Massachusetts at this time not more than 25 or 30 of these particular cases) present quasi-normal appearances that have been described sometimes as psychic malformations, and often give the impression of ability to fit in to a reconstructed world, with the patients in the center as kaisers or I. W. W. chiefs therein. In short, many of these paranoiacs, victims of the severest and most intractable psychosis known, are nevertheless without a trace of mental splitting such as I have hinted at above. And in fact the paranoiac in question showed no signs of mental dissociation, but rather showed signs of what might be termed a hypersynthesis of such facts in her environment as fitted her morbid ego and her jealousies. What I am getting at is that the psychopathological analysis of this case of paranoia precisely did not demand any evidence of supracortical disease; in fact, had there been supracortical disease of any moment we should have wondered whether there had not been some disintegration of the patient's personality, at any rate at the close of her life. It is interesting that this particular paranoiac did show some infrastellate disease which, as will be mentioned below, I regard to be correlated with auditory hallucinosis which she showed.

The other three cases intensively examined (I may recall that notes were made as to different types of cell loss, gliosis, satellitosis, vascular changes, etc., for each layer in each of 25 to 30 areas of the cortex in each subject) showed signs of supracortical disorder as well as of infracortical disorder of varying degrees in different parts of the cortex, and all three of these cases showed signs of schizophrenia. This evidence is at least suggestive that supracortical disorder is necessary in schizophrenia. I felt satisfied in my own mind, from the results of the work, that not only is the cerebral cortex the proper study of the psychologist and the psychiatrist, but that the supracortex is in a still more intimate sense the proper study of the psychologist or psychiatrist who has to deal with the higher functions of what may be called psychic association or synthesis. To my mind such studies, if in future confirmed, will throw an important added light upon the problems already partly illumined by comparative anatomy.

Let me insist here, if it is not superfluous, upon the fact that I am not proposing an hypothesis to the effect that every time a mental split occurs a cell drops out of the supracortex. I do not even see in my mind's eye a dendrite dropping off of a supracortical cell to signalize the cleavage of an idea. There is very properly much fun to be made of workers who regard ideas as fine transparent substances poured in and out of nerve cells as wine in and out of goblets. Aside from the problem of parallelism and interactionism, suggested by the figure that regards brain cells as vehicles for ideas, my own contentions deal rather with the observational facts. I would regard all gliosis as merely indicating and not defining the type of cortical disease in question, and I should regard the nerve cell losses or other changes as likewise nothing but indicators of the kind of thing going forward, or perhaps as indicators that something or other is going forward in the injured tissue. To be sure, I would think that destructive brain changes would be more likely to be associated with mental dissociation (with schizophrenia, gliosis) than with other types of synthetic or redistributive mental processes. Supracortical neurone psycholysis, to put the whole matter in a formula, is to my mind correlated with schizophrenic processes. What, you may well ask, is to be correlated with hypersynthetic, falsely synthetic or redistributive mental processes of the mind-twist sort? For the present I, for one, have no idea what cortical process corresponds to these non-destructive morbid psychic processes. Mind lack and mind loss should correspond with the cell lack and cell loss.[2] What we term figuratively mind twist, *e.g.*, false reasoning without loss of sensory intake and power, without loss of memory images, without loss of power of expression, without any evidence of coarse loss of function, remains as a process not readily statable. Perhaps we must fall back here upon the idea of physico-chemical changes and of anomalous distributions of energy in normal neuronic systems. But these are speculations so remote from observational facts that so far as I can see they cannot do the observer any harm.

Besides schizophrenia there are other bizarre effects in dementia præcox. Perhaps the strangest thing in the world is catalepsy. Catalepsy, catatonia, cerea flexibilitas are not infrequent symptoms in many cases of dementia præcox. They all have the character of muscular hypertension somewhat suggestive of hypnotic effects, or, again, of the effects of drugs upon the muscular system. Sometimes the phenomenon of negativism and resistivism remains one of the Sherringtonian experimental effects showing innervation of antagonistic muscle groups. Possibly these processes are to be explained upon some general line as schizophrenia; possibly they are due to some kind of lysis;

some workers are inclined to show the concept of inhibition very freely in the attempt to explain them. They are often a transient phase recurring again after comparatively normal intervals in a schizophrenic patient, and it seems impossible to evoke them by any form of stimulation of the patient.

In a general way such conditions as catalepsy seem a good deal more like phenomena produceable in lower animals than would schizophrenia. Some of the hypnotic or pseudo-hypnotic appearances in lower animals roughly resemble the catatonic effect. It was in a case of catatonia that Alzheimer first stated that there were nerve cell changes in dementia præcox, and it will be remembered that he placed these changes (chiefly gliosis) in the lower layers of the cortex. To a certain extent, my own investigations here confirm those of Alzheimer; at all events, in the case showing catatonia there were lesions in the infracortex.

Another symptom not at all so bizarre as schizophrenia and catatonia is hallucinosis. Hallucinations are rather quasi-normal effects that the normal person rather readily understands and that occur in a great number of mental diseases. Hallucinosis is in short a far less pathognomonic symptom for dementia præcox than is either schizophrenia or catatonia. Many forms of hallucination seem to be of so simple a nature that they could well be elicited in the lower animals could we only get at the psychic interiors of the lower animals to learn their mental contents. *À priori,* therefore, it seemed to me that the disease process underlying hallucinosis ought rather to be in the infracortex than in the supracortex, and there was some confirmation of this idea in the case studied. I mentioned above the fact that late in the disease the paranoia patient developed auditory hallucinosis. She showed infracortical cell loss of a mild but distinct degree. It will be remembered that the brain of this case was the most nearly normal of all the brains so far studied in the whole series.

The stratigraphical analysis of these brains proved, accordingly, very alluring. I seemed to get evidence that the supracortex was a region whose disease might well spell schizophrenia, whereas the infracortex was a region whose disease was related with lower level symptoms, such as catatonia and hallucinosis. The supracortex and the infracortex would then be regions of differential interest for the psychiatric investigator who would seek in the supracortical region for evidences of higher intellectual disorder in the field of association combination and abstraction, and in the infracortex for evidences of lower forms of mental disorder, such as catalepsy and hallucinations. I would not stick on the question of higher and lower functions at this point. Catatonia may well be a far more complex matter than many forms of intellectual dissociation. I call it lower because it seems to me that the simpler organism could show catalepsy rather than disorders of combination and abstraction in the intellectual field.

I have latterly reviewed the cell findings in the different cortical areas, assuming that I should find rather suggestive topical correlations between special kinds of symptom and special loci. I assumed that *à priori* no one would be likely to hunt for the source of auditory hallucinations in the small zone as defined by the comparative anatomists, and that one would hardly be likely to look for the basis of visual hallucinations in the superior temporal gyrus.

Perhaps, in accordance with the opinion of the learned psychopathologist above mentioned, it is *à priori* certain that in point of fact auditory hallucinations have their basis somewhere in the temporal lobe, and visual hallucinations somewhere in the occipital region, and why undertake a superfluous task of setting out exactly where and under what conditions they occur? We ought to be greatly helped in this matter by the focality of lesions in dementia præcox.

In the beginning I had been attracted by the fact that there was often a lobar or lobular hypoplasia and atrophy or sclerosis in these brains. In fact, I had tried reasoning from the gross data alone to draw tentative conclusions as to the main lines of functional differentiation in the symptoms of dementia præcox.

Noting how difficult was the distinction between inborn anomaly and acquired lesion, and how uncertain one might be that a given hypoplasia had anything to do with a given disorder of function, I felt that I ought, perhaps, to investigate cases without gross lesions.

Now one of the most generally significant results of this intensive examination of numerous areas and all layers in dementia præcox brains is that there is a certain focality in the microscopic lesions. I still find the focality to be rather a lobar or lobular one than an intragyral focality. To be sure, my colleague, Dr. H. I. Gosline,[9] has made some interesting observations upon some exquisitely focal lesions of intragyral distribution, possibly related with tuberculosis in certain dementia præcox brains. Gosline has endeavored to show that the situation of these lesions (demonstrable by fat stains) is such as to fit with my own older contentions as to the relation of certain symptoms to particular regions. Notably Gosline endeavors to confirm my idea of the correlation of catatonia to post central lesions. My own work, as here reported, deals not with results of fat stains, but with the results of the study of cell losses by the ordinary tinctorial methods for determination of tubercle, at least with nerve cells (Cresyl violet after formalin fixation). I have tried to show the cell losses thus made out as a little more reliable than the fat-stained cells and deposits that Alzheimer, Cotton and Gosline have used. Using the methods of these observers I obtain too rich a display of lesions, as a rule, to permit correlation.

The kind of focality of lesions which I believe to have demonstrated in these brains is not intragyral, but lobar and lobular, or in certain cases a focality of lesions affecting the whole gyrus. By and large, in these brains as one examines the different layers within a gyrus, the layers are apt to show throughout the gyrus the same kind of lesions, whether gliosis or cell losses. In short, the focality of these lesions is not the focality of tubercles, tumor masses, glanders lesions or exudative lesions. It is rather a focality such that, *e.g.*, the post central gyrus on one side is affected in a certain way throughout the gyrus with such and such layers homogeneously affected. The post central gyrus of the other side is relatively normal.

What now are the functional results of a study of the topographical distribution of the lesions just mentioned? Can we by focalizing attention on certain gyri discover functional correlations of lesions with certain symptoms?

The functional correlation of my dementia præcox studies, published in 1910 and 1914–15, were summed up from the topographical point of view as follows: —

1. Delusions are, as a rule, based on frontal lobe disease.
2. Catatonic symptoms are, as a rule, based on parietal lobe disease.
3. Auditory hallucinosis is, as a rule, based on temporal lobe disease.

Re the frontal lobe correlation with delusions, I found an exceptional group of delusional cases without frontal emphasis of lesions, but I found a plausible reason for these exceptions. Upon analysis the non-frontal cases of delusion formation turned out to be cases with a tendency to what may be termed hyperphantasia, that is, to an elaboration of fantasies not more than half believed, or at any rate not fully believed, by the patient. It is clear that from what we know of the probabilities of localization in the brain, both from a comparative and anatomo-clinical point of view, the parietal lobes might plausibly be implicated in the function of imagination, and that parietal disease might well be attended with functional disorders of imagination, such as overimagination, or what is here termed hyperphantasia.

It is plausible to argue that the parietal tissue, newly evolved in the higher animals, lying between the tactile post central region and the visual occipital region and the auditory temporal region, should be a tissue related with apparatus for combining these sensory data, — a tissue which might unobjectionably be termed a center for combining percepts and concepts involving two or more forms of sensation or perception. Whatever the merits of this *à priori* contention, at all events I found a tempting correlation of fantastic delusions with parietal lobe atrophy. I summed up the anatomical situation as follows: —

On the whole, the correlation between delusions and focal brain atrophy (or aplasia capped by atrophy?) is very strong, particularly if we distinguish (1) the more frequent form of delusions with frontal-lobe correlations from (2) a less frequent form with parietal-lobe correlations.

The non-frontal group of delusion-formations the writer wishes to group provisionally under the term "hyperphantasia," emphasizing the overimagination or perverted imagination of these cases, the frequent lack of any appropriate conduct-disorder in the patients harboring such delusions, and the *à priori* likelihood that these cases should turn out to have posterior-association-center disease rather than disease of the anterior-association-center. This anatomical correlation is, in fact, the one observed.

My previous work had also suggested a possible correlation between catatonic phenomena and parietal (including post central disease). Ten of fourteen definitely catatonic cases yielded gross lesions in the parietal or other post-Rolandic regions. Of the remaining four, two, negative in the gross, proved to be microscopically altered in the parietal region, and there were indications of a correlation in the remaining two cases. It must be remembered with respect to correlation that I am using the term "catatonic" here as the name of a symptom. I am not offering a correlation between anatomical or microscopic lesions and the catatonic form of dementia præcox.

For example, the symptom cerea flexibilitas is not necessarily an indicator that the victim belongs in the so-called catatonic form of dementia præcox, despite the fact that cerea flexibilitas is one of the prettiest examples of catatonia ever described. Four of the five cases in the 1914–15 series that showed cerea flexibilitas had parietal anomalies or atrophies, and the fifth case, though it was entirely negative in the gross, was a case which showed an extreme degree of satellitosis in the post central region examined microscopically. These observations and certain *à priori* considerations led me to formulate the idea that catatonia, and particularly cerea flexibilitas, was a form of disorder of kinæsthesia.

Re the correlation of auditory hallucinosis with temporal lobe lesions, it is easy to see that such a correlation ought to exist if any gross correlations at all were expected. In point of fact, nine of my twelve hallucinating cases have had temporal lobe atrophy or aplasia. One of the three cases had ample microscopic changes in the temporal lobes, one was clinically somewhat doubtful, and in fine, only one of the twelve hallucinating cases could be safely said to have neither gross lesions nor important microscopic lesions in the temporal area.

To sum up, then, we expect frontal lobe lesions in cases of delusion formation, provided that the delusion formation is not of the fantastic and overimaginative sort, whereupon parietal lesions would be expected. We expect parietal lesions associated with catatonic symptoms; we expect temporal lobe lesions in cases of auditory hallucinosis.

Accordingly, I have collected the microscopic data in the four cases at present under discussion under these heads, — frontal, parietal and temporal. For convenience I have divided the cortex into three portions, — frontal, parieto-occipital and temporo-occipital. The following table indicates the extent of nerve-cell loss and of neuroglia proliferation separately in the three regions mentioned: —

	I (10.9), 14 Years.	II (12.41), 10 Years.	III (12.47), 2 Years.	IV (11.36), 20 Months.
Frontal loci,	9	9	9	6
Nerve-cell losses,	5	44	35	34
Neuroglia proliferation,	7	8	4	3
Parieto-occipital loci,	10	9	10	7
Nerve-cell losses,	17	47	54	19
Neuroglia proliferation,	8	3	2	8
Temporo-sphenoidal loci,	10	10	10	6
Nerve-cell losses,	9	48	50	25
Neuroglia proliferation,	5	9	1	-

It is clear from inspection of the table that Case I (10.9) stands out as a case in which the parenchymal disease is especially parieto-occipital, since in ten loci, seventeen instances of nerve-cell losses in different layers were found. Here, then, should be a case with catatonia, unless, perchance, the lesions in the parieto-occipital area should be exquisitely occipital and not affect kinæsthetic areas. In point of fact, the lesions enumerated are all in the post central, superior and inferior parietal areas, and are especially marked in the inferior part of the post central gyrus and in the superior parietal region on both sides.

This patient was one who developed characteristic catatonic symptoms, including cerea flexibilitas and attitudinizing in the last two years of her life. She also had auditory hallucinosis and showed, as the table indicates, a number of parenchymal losses in the proper area, especially the superior temporal gyrus. Both the parietal and the temporal lesions were largely infracortical in distribution, in accord with the argument mentioned above as to the infracortical origin of catatonia and hallucinosis. It will be noticed from the table that there were comparatively few lesions in the frontal region. The case was one of delusions of jealousy, and might have been expected to have shown frontal lesions which, however, were not greatly in evidence. However, upon closer analysis of the delusions it will be found that they are of a synthetic character and not of a schizophrenic character. They are quasi-normal, and it is not easy to conceive just what histological basis such delusions ought to have. At all events, none of any consequence were found, and the frontal region remains practically destitute of any parenchymal loss, and has exceedingly slight evidence of gliosis. Accordingly, we may suppose that we are getting histological evidence in this case merely of the terminal hallucinosis and catatonia, and not of the essential paranoia of the patient.

If we turn to Case II (12.41) we find a totally different situation. The amount of parenchymal loss, as indicated by the number of loci, is far larger than in Case I (10.9). In fact, there are four times as many loci affected, and the lesions are scattered throughout all three regions, — frontal, parietal and temporal. There are relatively more parenchymal losses in the frontal region than in the temporal region, and relatively more in the temporal region than in the parietal region.

If we took these data on their face value we should say that this case ought to be predominantly delusional but also hallucinatory and catatonic. There was some question whether the patient was not slightly feeble-minded to start with. However, her command of theological verbiage was large, and she was always self-supporting up to the time of her psychosis. My point in mentioning this query is that very possibly some of the parenchymal losses are actually due to an initial lack of cells.

The interpretation of an initial lack of cells in any of these areas would of course look in a different direction. An initial lack in frontal cells might not at all suggest delusional tendencies; an initial lack of superior temporal cells likewise might have nothing to do with auditory hallucinosis; and a simple parietal mechanism would not necessarily entail disorders of kinæsthesia with the projection of catatonia. But taking the parenchymal deficiency as signifying initial loss, let us inquire what the symptoms of this case are. Was it predominantly delusional? She showed hallucinations of a doubtful but probably hypnogogic visual nature. It is not clear that she ever had auditory hallucinations. The appearance of parenchymal loss has proceeded in all these areas to a marked degree, so marked, in fact, that it is rather surprising that there was no gross evidence of brain atrophy (the brain weighed 1,130 grams, possibly a slight reduction from the normal weight; the patient was cardiac, and there was such a condition of infiltration of the brain tissue that we can *post hoc* readily believe the brain was slightly and diffusely swollen). In short, the lesions in this brain are so numerous and widespread and so affect both the supracortical and the infracortical regions that correlations of the nature here attempted are impossible. One can find a correlation between almost any symptoms and some properly situated lesion in this case. The patient was deluded, hallucinated and catatonic, and had frontal, temporal and parietal lesions.

Let us, for the sake of argument, regard the frontal lobe delusion correlation as established; let us regard the parieto-occipital correlation with "feelings of being hypnotized;" and the "black cat's jumping over a pitcher" as indicators of disease in the parieto-occipital or occipital areas. How, then, shall we interpret the absence of auditory hallucinations? Without invoking the poverty of clinical observation, and the fact that the patient may have kept auditory hallucinations to herself (she was, in point of fact, often engaged in vituperative language, some of which may have been of hallucinatory origin or of a responsive nature), we must be interested in the fact that some of the temporal lesions shown are exceedingly heavy, and affect both the supracortex and the infracortex. Suppose infracortical, temporal lobe lesions of a nature suitable to produce hallucinations (we are still ignorant just what sort of lesion ought to produce hallucinations), would it be too speculative to suggest that the co-existence of severe supracortical lesions might still interfere with the proper combination and abstraction of the infracortical data in such wise as to produce conscious hallucinations? That is to say, the total supracortical and infracortical mechanism may be conceived to be so altered that though the conditions for hallucinosis are present in the infracortical zone, the hallucinations are not purveyed through lack of finer connections with the higher psychic apparatus mediated by the supracortical layers. Naturally I do not wish to claim more than speculative value for such considerations. It is along these lines, however, that I feel much important correlation will be performed in future.

If we inspect the table of local distribution of lesions in Case III (12.47) we find conditions approximating those of Case II (12.41), although the duration of Case III is far less than that of Case II. We find here that the parieto-occipital region has numerous lesions slightly in excess of lesions, also numerous, in the frontal and temporal areas. The case was a catatonic one, and, in fact, showed both cerea flexibilitas and catatonic stupor, and would probably be classified as a case of catatonic dementia præcox. It appeared that she had auditory hallucinations which she tried to keep out by covering her head with her arms, but the content of these hallucinations was never made out. An attitude of apprehensiveness, an early phase in which she constantly repeated the words "Father Patterson," "Father Patterson," led us to the idea that she had delusions of persecution, or else that she was extremely apprehensive and threw a delusional covering over everyday features of the environment. The most pronounced regions of cell loss were in the infracortical portions of the parietal and temporal regions.

When we come to the analysis of Case IV (11.36) we find that the parieto-occipital lesions are far fewer in number, and that the case is more one of losses in the frontal and temporo-sphenoidal areas. This case, therefore, should show delusions and possibly auditory hallucinations. She never showed convincing catatonic symptoms, although she showed a marked mania and hyperkinesis somewhat suggestive of manic-depressive psychosis. She showed characteristic auditory hallucinosis, and it appears that most of her anti-social acts of a mischievous and silly nature were based upon these hallucinations. That she showed definite delusions, *e.g.*, of persecution or of poisoning, is doubtful. We know little of her history immediately after the onset of mental symptoms which followed confinement in a hospital for consumptives. Throughout her period of observation in a hospital for the insane she showed elation, erotism, mischievousness and silliness somewhat suggestive of manic-depressive psychosis, and again somewhat suggestive of the elation of certain paretics. The cell losses in frontal region were numerous and both supracortical and infracortical in distribution. It is interesting that of all four cases this one was the case which showed a marked myelin sheath disorder, — a disorder so marked as somewhat to suggest the disappearance of fibers in paresis. The supraradiary fibers were absent in all the frontal regions examined, and there was a thinning of fibers in the white matter underneath. In short, the disorder in this case was much more than a fine disorder of minor cell mechanisms, and approached the more general disorder of paresis. Upon some such lines I would be tempted to try to explain the absence of definite delusions, except those based upon hallucinations and irritability. I should be tempted to regard the frontal lobe lesions as of such degree that we might think of the case as one that had lost its frontal inhibitions. At the same time, it must be remembered that no coarse atrophy of the brain had supervened, unless we regard the brain weight — 1,140 grams — as demonstrating brain atrophy.[1]

SUMMARY.

Thanks to the work of Elliot Smith, Bolton, Campbell, Brodmann, Ramon y Cajal and others, the neuropathologist can now afford to attempt finer functional correlations in the field of mental diseases, thus aiding in the problems of microlocalization. The anti-localization tendencies of the Wundtians, and the interest in merely logical categories taken by Freudians, should not interfere with progress in microlocalization. Dementia præcox, for example, can be called a matter of maladaptation of the patient to his environment, or of the patient to himself, and also a disease characterized by cortical changes.

Previous work had shown anomalies in a high proportion of dementia præcox brains, and in a corresponding low proportion of the brains of manic-depressive subjects. These anomalies may well be interpreted as weak places in these dementia præcox brains, and the brains, in fact, are apt to show scleroses and atrophic processes over and above the anomalies. But certain perfectly normal-looking brains in dementia præcox also show the same microscopic changes in lesser degrees than are found in the anomalous sclerotic and atrophic brains. The problem of the present communication has been to work out the focality of these microscopic lesions in a few normal-looking brains studied with unusual intensiveness. In the same series of brains, work of previous series had shown a dissociation of parenchymatous (neuronic) and interstitial (neuroglia) changes, indicating a tendency on the part of cortex pathology to resemble the pathology of the kidney. But the majority of brains show mixtures of the parenchymatous (neuronic) and the interstitial (neuroglia) lesions. Recent work in comparative anatomy indicates the rather fundamental importance of distinguishing the functions of the upper cortical layers (what may be called the supracortex) from the functions of the lower cortical layers (what may be called the infracortex). The finer processes of mental dissociation (schizophrenia) ought to be correlated with lesions of the supracortex, and such lesions were found in cases with evidence of schizophrenia.

On the other hand, in a case of delusions characterized by no splitting (schizophrenia) whatever, but rather by a process of overelaborate synthesis, there was no evidence of supracortical disorder, and, in fact, no proposal can be made for the histological correlate with this process of oversynthesis.

Other processes equally characteristic of dementia præcox, but logically far simpler in their make-up, such as auditory hallucinosis and muscular hypertension (catatonia), received suggestive correlation with processes in the lower layers of the temporal and parietal regions, respectively.

As far as the tissues of these four cases go there is little or nothing inconsistent in the finding of the hypothesis that ordinary (non-fantastic) delusions are correlated rather with frontal than with otherwise situated lesions; but the supracortical type of delusions found in certain long-standing paranoiacs, whose fine mental processes run in a quasi-normal manner, find no special correlation in any region, and the probable importance of the solution of this problem remains obscure.

As for auditory hallucinosis the work seems to afford the expected correlation with temporal lesions. In one case, however, temporal lesions of considerable severity were attended in life by hallucinations of hearing, but in this case there was also a severe supracortical disease of the temporal region, and it may be that for the production of hallucinations some congress is necessary between the activation of the supracortex and infracortex, respectively.

In previous work on this series the brains had indicated a post central and superior parietal correlation for catatonia whose muscular hypertension was accordingly regarded as very possibly a kind of morbid kinæsthesia. Present work suggests that the anatomical correlate is not merely to the post central and parietal regions, but still more specifically to the infracortical parts of these regions.

It may be suggested that the lesions found in samples of tissue in the post central, superior parietal, inferior parietal and superior temporal regions indicate a certain systemic tendency in the underlying processes, for these lesions were bilateral, and occurred, as it were, in two continuous sheets of tissue on both flanks of the brain in one of the best defined of our cases; these flank lesions were not attended by any similar lesions of the frontal, precentral, occipital and lower temporal and smell regions. The nature of a process which could mildly affect nerve cells and neuroglia on two sides of the brain, and also specially affect the infracortical rather than the supracortical portions of these affected sheets of tissue, remains a mystery. It is perhaps no greater mystery than that which attends the distribution of lesions in the spinal cord of pernicious anemia, or the fine symmetrical lesions of multiple sclerosis in the spinal cord. It remains unsettled whether these lesions are secondary in point of time to a non-cell-destructive phase in the disease, or whether the lesions of which these microscopic effects are indicators began *pari passu* with the symptoms; that is, it remains a question whether we are dealing with the excess wear-and-tear process of cell mechanisms morbidly employed, or whether the morbidity of neural function is an exact equivalent of the neuronic and neuroglia morbidity.

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THE DIFFERENTIATION OF PSYCHOTIC FROM NEUROTIC CONDITIONS.*

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In Tuke's "Dictionary of Psychological Medicine," published in 1892, are found these definitions: "Psychosis, — a mental affection; psychoneurosis, — a mental disease."

To be sure, these definitions are many years old, and since then the situation has been somewhat clarified, but to many practitioners and laymen the clinical differentiation of psychotic from neurotic conditions remains about as nebulous as the above-quoted definitions.

A part of this vagueness of understanding has been due to the relationship between psychoses and insanity, but this difficulty is resolved by recognizing that insanity is a social condition, — a question of conduct in a given environment, and a question primarily of legal rather than medical definition. It need hardly be stated that many people are adjudged insane who have no psychoses, while many psychotic individuals are never judged insane. Many a wild hysteric has been committed as insane, and rightly, because of his or her conduct, but not because of suffering from a psychosis; and many a senile dement has been lovingly cared for at home as a sweet old grandmother. Clinically, we are familiar with the diagnosis of pre-dementia præcox and paresis sine paresi in individuals whose conduct does not at the time warrant commitment as insane.

The matter of insanity, then, must be considered apart from that of the existence of a psychosis, for insanity, as has often been stated in court, can be described but cannot be defined. In fact, Lord Justice Blackburn is quoted by Church and Peterson as saying, regarding insanity: "I have read every definition which I could meet with, and never was satisfied with one of them, and have endeavored in vain to make one satisfactory to myself. I verily believe that it is not in human power to do so."

The differentiation of a psychosis from a neurosis is, however, a matter of prime importance and worthy of discussion. To the layman a vast gulf divides the psychotic from the "only nervous." He will go to the psychopathic hospital readily, but cannot be dragged to a State institution. He will go to a nervine hospital, but hesitates even to pass by a sanitarium for mental cases. His sick neighbors are perhaps at times insane, but his own relatives are only "very nervous." He will consult a neurologist, but hesitates to call on a psychiatrist. The family practitioner prefers to consider a case "nervous," whereas the psychiatrist may often remind one of the old Quaker who is famed for saying, "Every one is queer but me and thee, and sometimes thee is a little queer."

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* Read at the meeting of the Boston Society of Psychiatry and Neurology, Oct. 18, 1917.

Surely some clinical standard should be found that will help clarify this situation.

In alcoholic intoxication the law has recognized the distinction between delirium tremens and alcoholic hallucinoses, and provides for the exclusion of the former and the admission of the latter to the Boston Psychopathic Hospital.

Kræpelin has given a table for differentiating these two conditions, in which ten factors are considered, as follows: Lucidity and orientation are absent in delirium tremens, present in alcoholic hallucinoses. The sensorium is clouded in the former, clear in the latter. Hallucinations are largely of sight in tremens and of hearing in alcoholic hallucinoses. Suggestibility is increased in the former, normal in the latter. Restlessness is increased in tremens, absent in the other condition. Consistent delusions are absent in tremens and present in alcoholic hallucinoses. Coarse tremor is present in the first condition, absent in the second. The course of the disease is rapid in tremens and slow in alcoholic hallucinoses, and the termination of the former is by crisis and of the latter by lysis.

In other words, as a general thing, in delirium tremens there is evidence of a toxic interference with the receipt of stimuli and an increased reaction through the vegetative nervous system to emotional stimuli, whereas in alcoholic hallucinoses the evidence of interference with the receipt of stimuli is seemingly lacking, and instead of an increased reaction through the vegetative nervous system to emotional stimuli there is often a striking lack of such reaction.

A similar distinction can be seen in cases where another toxic agent is at work, namely, syphilis. In pre-paretic cases the symptoms suggest neurasthenia, and exhibit an increased reaction through the vegetative nervous system to emotional stimuli, whereas in full-blown paresis there is again a striking lack of such reaction.

In addition to alcohol and syphilis a third agent that reacts upon the body with symptoms varying through the whole gamut of neurotic and psychotic conditions is one upon which much interest and investigation is now focused, namely, shell shock.

Here, then, are found a chemical agency, a microbic agency, and a purely physical agency, each of which produces in man symptoms varying from mild neuroses to severe psychoses.

Study of this new agency, namely, shell shock, promises new and invaluable knowledge in the fields of physiology, and especially psychiatry.

In a recent article on "The Psychology of Fear," Sir Robert Armstrong-Jones has written most illuminatingly upon the subject. At the risk of boring those of you who have read his article, a short abstract of some of the knowledge and theories that he has presented will be given. He states "that in every emotion there is a cognitive and active and an affective experience, and that the emotions are the expression of — or, according to some, are themselves expressed in — characteristic instinctive acts." Fear, which in its various forms is a universal and fundamental human experience, is expressed physiologically in varying degrees by inhibition of all visceral movements, contraction of the blood vessels, lowering of surface temperature, sweating, cessation of the flow of saliva, dilatation of the pupils, erection of the hair, increased cardiac and respiratory activity, and tremblings and twitchings of muscles, especially of the face. In addition to these direct emotional reactions, through the vegetative nervous system, there is an indirect action through stimulation of the adrenal glands that results in an increased sugar excretion from the liver into the blood for the use of the muscles, a diversion of blood from the abdominal viscera into the heart, lungs and central nervous system, an increased coagulability of the blood, and an increased blood pressure. This adrenal secretion augments the effect of the emotions, and also helps to prolong and sustain it, and may be likened crudely to the ignition in an automobile, which at first is accomplished by the battery, and then prolonged by the action of the magneto.

Such, according to our present knowledge of physiology, is the mechanism of the relationship between emotional activity and physical reflex action.

In shell shock it has been suggested that, owing to the intensity of the emotional stimuli, a dissociation of the cerebro-spinal and the vegetative nervous systems takes place, resulting in uncontrolled and unregulated action of organs innervated by the autonomic system.

The following thesis is advanced for consideration. In the neuroses, neurasthenia, psychasthenia, hysteria, etc., there is a hyper-reactivity through the vegetative nervous system to emotional stimuli, whereas in the psychoses there is a hypo-reactivity through this system to such stimuli.

The following observations and cases are quoted to illustrate this thesis: —

In the syphilo-psychoses (the mental disease groups which have been suggested by Dr. Southard will be considered) there are recognized the syphilitic neurasthenics, the pre-paretics, the cases of paresis sine paresi and the paretics.

Case A. — A case of neurasthenic syphilis, who complained of being "all in." "Could not think." Did not want to go home to his family, too nervous to stay at his office. Admitted luetic infection several years previous, and had had thorough treatment under skilled hands. Physical examination showed increased reflexes, healed ulcers over the shins, and a general hypersensitiveness to emotional stimulation. Appetite and sleep poor. Bowels sluggish. With rest, baths and psychotherapy he gained in weight and poise, and has been able to conduct his business for several years with marked success.

Case B came to the out-patient department of the Psychopathic Hospital because when he got out of bed he could not put one foot readily into his slipper. His speech was slightly thickened and his pupils sluggish and irregular. He was referred to the hospital for observation, where all laboratory tests were found positive for paresis.

Case C complained of inability to concentrate his mind. Did not feel well. Worried over an inability to pronounce certain words clearly. Was restless, anxious about every physical symptom. Physical examination showed almost absent knee jerks, irregular pupils that usually failed to react to light. Laboratory tests were positive for general paresis. With intravenous salvarsan, baths and psychotherapy he improved in physical condition, and seemingly became able to enunciate more clearly. He lost many of his worries and returned home in much better shape. When last seen he was not committable, and could scarcely be classed as psychotic, although from the laboratory point of view a clear case of paresis.

A typical case of paresis need not be cited, as the changed affectivity is recognized as an early symptom almost always present. The common immunity of the paretic to unpleasant after-effects from lumbar puncture is recognized, and when a case has nausea, headache or dizziness after a lumbar puncture a positive Wassermann reaction is not expected.

In the hypo-phrenoses — the feeble-minded — keenness of reaction to emotional stimuli is not expected. In case of fire at a feeble-minded school it has been remarked that the patients are less liable to be panic-stricken than the nurses and attendants.

Case D. — A young man seen in the out-patient department of the Psychopathic Hospital. He was so cheerful and well nourished and emotionally placid, in spite of a series of major operations of doing and undoing a gastro-enterostomy, repairing a hernia, and breaking up abdominal adhesions, performed at the Massachusetts General, the Carney, and the Brigham hospitals, that he was given a Binet test and proved to be but nine years of age mentally.

Case E. — A high-grade moron of gluttonous habits, who insists on constantly overeating. Often after excessive eating he will easily regurgitate part of his meal with as little discomfort as an overturned bottle.

Among the epileptoses a change in affectivity is noted that may gradually develop and remain present, or may precede or follow individual seizures.

Case F. — A woman, eighty years of age, who since childhood has had epileptic attacks varying in frequency from none to fifteen a week. Within the past twelve years she has gradually developed a psychotic condition, and now shows almost complete lack of memory, and at times confusion and irritability. Her mother, herself, her daughter and her grandmother have suffered from epilepsy. Now that her psychotic condition has developed she is placid and cheerful most of the time, going driving daily, and has shown practically no physical enfeeblement within the past five years.

Among the pharmaco-psychoses — the alcoholic and drug cases — the distinction between delirium tremens and alcoholic hallucinoses has already been considered.

Among the encephalo-psychoses — the coarse brain disease cases — are to be found cases that in their early stages are classed as neurasthenics that later show marked changes in affectivity.

The somato-psychoses are seldom seen in their initial stages by psychiatrists. Experienced general practitioners, however, recognize in typhoid, cardiac, pneumonic and puerperal cases a change in affectivity, a keen alertness, a disregard of normal physical weakness and disabling symptoms that put them on their guard to prevent sudden elopement or tragedy. Osler states in his "Practice of Medicine" that acute mania occurs as a complication of various somatic conditions, but gives no hint to assist a practitioner in recognizing the onset of such a condition.

Case G. — A case of marked cardiac decompensation. If unwatched he would spring out of bed and run out of his room, and show evidence of strength and agility absolutely incompatible with his physical condition.

Among the gerio-psychoses are again to be found cases varying in symptoms from neurasthenias to frank psychoses. Affectivity changes are an early symptom of psychotic change, and the virility and endurance of senile dements often far surpass that of non-psychotic elders.

Case H. — A woman of nearly eighty years, blind, deaf, toothless and partly crippled with arthritis, has often required two or three nurses to hold her when her nails have to be manicured.

Case I. — A man of seventy-five, who had not eaten of his own volition for months, requires two strong male nurses and a doctor when he is tube fed.

Among the schizo-phrenoses — the dementia præcox cases — change in affectivity is an early and almost constant symptom. In fact, it may be suggested that "the shut-in personality" may possibly better be thought of as a "shut-off personality," an individual in whom the connection between emotional stimuli and normal reaction through the vegetative nervous system is irregular and labile, predisposed to rupture under environmental or physical strain.

Case J. — A woman of thirty-five, a dementia præcox case of several years' standing, who required the removal of several old teeth. She was seized, heavily etherized, and had four teeth extracted. When seen a quarter of an hour later she was sitting on the edge of her bed, asking to go out into the garden, and showing no nausea or evidence of shock from her uncomfortable experience.

The cyclo-thymoses — the manic-depressive cases — also show types varying in symptoms from mild neurasthenic-like depressions to frank psychoses.

These cases imperatively call for an early diagnosis, for from among these cases come the sudden suicides and maniacal murders. When the hard-working business man or military officer, with heavy cares and responsibilities, ceases to be fatigued by excessive labor, craves more work, less sleep, and is indifferent to nourishment, danger should be scented and relief from emotional strain secured.

Among the frankly maniacal cases, excessive motor activity, without apparent fatigue or need of sleep, is often marked. Exposure of the person to cold and wet seldom, if ever, results in colds or reflex congestion of the nasal mucous membranes.

Case K. — A woman of sixty, who had usually suffered from frequent colds and so-called "hay fever," upon the development of a prolonged agitated depression ceased to have any signs or symptoms of such vasomotor disturbance. She also failed to show any nausea or evidence of shock after being heavily etherized.

Case L. — An elderly woman who had suffered for years from asthmatic attacks ceased abruptly to have these attacks subsequent to the onset of a long depression.

Among the psychoneuroses there is universally recognized a relationship between emotional disturbances and the physical symptoms that, to a greater or less degree, incapacitate them for effective life and happiness. In the neurasthenias these symptoms seemingly represent excessive reactions through the vegetative nervous system in the field innervated thereby, — the digestive system, the urinary organs, the genital organs, the respiratory apparatus, the cardio-vascular system, etc., while in hysterics and shell shock cases there is seemingly an overflow of reflex stimulation that, in addition, inhibits various muscular activities usually controlled through the cerebrospinal system.

The scope of this paper does not permit of a discussion of the etiology of the psychoneuroses. That the emotions play an important part in these conditions is generally admitted. That the emotions react physiologically through the vegetative nervous system is also admitted. That recognition by a patient of this reaction through the vegetative nervous system gives rise to secondary emotions, and that these secondary emotions start a secondary reaction, and so on, with the formation of a vicious circle that becomes more and more complex the longer it spins, is also recognized.

Whether the genesis of the emotional disturbance is due to intra-uterine or extra-uterine sex trauma, to buried complexes, or due to congenital somatic deficiencies; whether the type or intensity of the stimulus, or the congenital or acquired weakness of the patient is the determining etiological factor, — all this is aside from the scope of this paper.

Among the psycho-pathoses — the psychopathic individuals — there may be assumed to be a lability of connection between emotional activity and the vegetative nervous system that in the genius enables him to surmount difficulties with exceptional success, and disregard the distractions and environmental inhibitions of more common individuals, — a lability which in another member of the same family, with failure, predisposes to mental disease.

CONCLUSIONS.

No attempt has been made to define insanity, which may properly be considered capable of description, but not of definition.

No attempt has been made to consider the etiology of various types of psychoses.

The suggestion is made that clarity of diagnosis will be furthered and consequently great help rendered in prognosis and treatment if a clear line of demarkation can be drawn between the psychoses and the neuroses; that cases of whatever etiology — alcoholic, syphilitic, determined or undetermined — be considered neuroses so long as they show hyperactivity of reaction through the vegetative nervous system to emotional stimuli, but that they be considered psychoses when there is found to exist evidence of hypo-activity of reaction through the vegetative nervous system to adequate emotional stimuli.

THE TREATMENT OF EARLY SYPHILIS IN RELATION TO THE DEVELOPMENT OF NEUROSYPHILIS.*

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Modern work on the treatment of general paresis, such as that of Solomon at this hospital, has clearly shown that there are cases which for some as yet unknown reason do not improve; or, if they improve, relapses are early and marked. Similarly there are cases in which vigorous treatment in the early stages of syphilis will not prevent the later development of paresis.

The difficulty of obtaining from cases of neurosyphilis accurate histories regarding syphilitic infection and symptoms is well known. Still less often are we able to obtain any accurate information regarding the precise amount and character of antisyphilitic treatment at the time of infection. Accordingly, the cases reported here should be of general interest because of the known amount and character of the treatment. A further interesting and suggestive point lies in the results of the type of treatment developed and applied by Solomon. This seems to me to carry a wholesome therapeutic lesson.

Case I. — A male, age 28, came voluntarily to the hospital Dec. 11, 1916, complaining of loss of memory, speech difficulty and irritability, all developed within the six months just past. He was tidy, elated, showed a mild memory defect, was somewhat silly, over-emotional; circumstantial; rather wandering in his attention, and had a speech defect. Physical examination was not remarkable except pupils unequal, irregular and react sluggishly; tendon reflexes hyperactive; no sensory disturbance or ataxia.

Six years previously he had a chancre. During five years he received almost continuous treatment with mercury by injection. He had two injections of salvarsan. He never received any potassium iodide. He never showed any secondary symptoms.

Laboratory Findings. — Blood and fluid Wassermann reaction positive. Spinal fluid showed: globulin, 3; albumin, 4; cells, 34; and a gold reading of 5555431000.

In twelve days he received four injections, totaling 2.3 grams of diarsenol. He was then discharged to the out-patient department, where treatment was continued, so that on Feb. 14, 1917, the Wassermann

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reaction on the blood serum was negative, and the spinal fluid showed: globulin, 2; albumin, 3; cells, 4; gold reading, 2222210000. In the meantime there had been great physical and mental improvement. At the present time he shows slight tremors; a little stumbling on test phrases, but his ordinary speech is clear and accurate; his memory is good; and his insight into his past illness is good.

This case presented a clinical symptom-complex typical of paresis in its early stages coming on six years after the initial lesion, and after five years of rather intensive treatment. He has shown marked improvement under the type of treatment which has produced the best results in paresis, but the favorable time for such intensive treatment was months or even years ago, before the changes in the nervous system had advanced to any degree of degeneration.

Case II. — A male, age 34, came voluntarily to the hospital May 7, 1917, complaining that for about six months he had been tired, irritable and nervous, and was getting worse. He also recognized a memory loss. He worked in a post office at night and had spent his days for two years as a medical student. Normally ambitious and a hard worker he had worried a great deal over his condition; had become emotionally unstable, crying readily, depressed and showing memory loss. Aside from exaggeration of the knee jerks, and a very slight speech defect, the physical examination was negative.

He had a chancre three years ago. After the secondary symptoms appeared he received thorough and intensive treatment with salvarsan, mercury and potassium iodide. Mercury and potassium iodide have been carried in courses up to the time of admission. Of late he has been troubled a great deal by headaches. A part of his difficulty in medical school, where he did not do very well, may have been due to the spirochetal invasion of the central nervous system, though undoubtedly much of it was due to his combinations of work.

Laboratory Findings. — Wassermann reaction on the blood and fluid positive. The spinal fluid showed: globulin, 2; albumin, 2; cells, 8; and a gold reading of 5554332100. Under intensive treatment of the usual type his mental and physical condition improved very markedly, and on July 21 the spinal fluid showed: globulin, 0; albumin, 0; cells, 0; gold, negative.

At the present time, aside from slight memory loss and some slowing in his work, his condition is very good.

Case III. — A male, age 43, was brought to the hospital by the police on Aug. 16, 1916. He was then in a highly excited state, grandiose and talkative. On examination he was oriented; active; euphoric; voluble; there was some flight of ideas; and he had no insight.

Physical Examination. — The pupils were unequal, irregular, Argyll-Robertson in type; the tendon reflexes were absent; there was some inco-ordination; he had a high blood pressure.

At twenty-seven, sixteen years ago, he had a chancre. During the succeeding ten years he was treated almost continually with mercury and iodide. The principal reason for this was the frequent recurrence of skin symptoms. When salvarsan was introduced he was among the first in Boston to receive it. With this treatment his skin lesions healed. Then for six years treatment was not so intensive. Despite all of this treatment, and the marked skin lesions (which are sometimes supposed to argue against the later development of neurosyphilis), he eventually developed paresis. Under intensive treatment he improved sufficiently to go on a visit in November, remaining out for a year. He was then returned in a maniacal state, having refused treatment for six months before readmission.

At the time of his first admission the laboratory findings were: blood and spinal fluid Wassermann reaction positive. The spinal fluid showed: globulin, 3; albumin, 2; cells, 8; and a gold reading of 5555432100.

Despite the clinical improvement under treatment the spinal fluid always remained positive to the various tests.

These cases do not add much to the solution of the important theoretical and practical question, "Why does any syphilitic have neurosyphilis?" but they do demonstrate the fact that, either because of special invasive powers or increased susceptibility of the nervous system, neurosyphilis may develop despite thorough remedial measures. They show very clearly that it is not a question of time interval after infection, or after cessation of treatment, since the interval between infection and paresis was three, six and sixteen years, respectively.

These cases bring sharply to the fore a question concerning the duty of the syphilographer to his patient. It is my belief that before being discharged from treatment, all syphilitics should have lumbar puncture to determine the condition of the spinal fluid. Certainly all patients who suffer much from headache, or show any neurologic or mental changes, however slight, should be punctured. If the fluid tests are positive, then treatment should be modified accordingly. Furthermore, if intensive treatment of the patient after paresis has developed will bring about an improvement, then intensive treatment is certainly indicated in all cases showing any changes in the spinal fluid, and the spinal fluid findings should be the guide in further treatment.

CONCLUSIONS.

Thorough early treatment is not a guarantee against later paresis. The time interval between infection and development of paresis is not especially modified by the usual treatment. Whether due to special invasive powers, lack of resistance in the nervous system, or drug-fastness of the organism, the fact remains that there are cases in which the most vigorous treatment fails to prevent the development of paresis, or even to delay it.

Patients in the secondary stages should be punctured, and treatment modified according to spinal fluid findings. If not all patients, then certainly all early syphilitics with headache or any nervous or mental symptoms should be punctured.

If intensive treatment will improve the patient who has developed paresis, then intensive treatment of the same type months or years before, guided by spinal fluid findings, should go far towards preventing the later development of paresis.

REPORT OF TWO CASES OF PARESIS WITH MENTAL PICTURE OF DEMENTIA PRÆCOX.

By Dr. H. L. Horsman,

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The following cases are unusual from the fact that they presented serological findings of paresis with mental pictures of dementia præcox. In fact, one was diagnosed as dementia præcox before Wassermann test was made on the blood and fluid, which test was made simply as a routine procedure and not because paresis was suspected in this case.

Case No. 1 (Miss I. S.). — This case is that of a young woman who was transferred to the Grafton State Hospital Nov. 25, 1914, from another State hospital whose abstract shows the following: admitted there July 5, 1914; female, age thirty-one; divorced (?); occupation, prostitute. Her history was very meager. Her father had lost track of her for twelve years till a week prior to admission. Her mother died when the patient was a baby, and her father married again. She was always a headstrong child, and always caused them trouble on account of her fondness for the opposite sex. When seventeen years of age she left home, going to New York, and lived the life of a prostitute. A week before admission her relatives were notified to come for her, this being the first news they had of her for almost eight years. She was brought to her father's home, and for several days was confused, talkative, hallucinated and in great fear. On admission she was almost mute, resistive and confused. The medical certificate on admission states that the eyes did not react to light, but examination at hospital showed them to appear normal. All reflexes were exaggerated, co-ordination was good and Romberg's sign not present. She remained catatonic all the time while there; was fed with a spoon; made irrelevant replies to questions; lay in bed without moving; was quite resistive and untidy.

Diagnosis. — Dementia præcox, catatonic. She was admitted to Grafton State Hospital Nov. 25, 1914, having been transferred from the hospital above referred to.

Examination showed the reflexes to be slightly exaggerated, with normal pupils. She had coarse tremors of eyelids and hands, no impairment of gait, and co-ordination tests were fairly well performed in spite of the fact that the patient was rather weak. She showed some swaying in Romberg position, also evident decrease of temperature-sensation of lower limbs.

Mental examination showed a young woman of slight stature, presenting a demented, disheveled appearance, and rather vacant facial expression, catatonic at times. She made no voluntary conversation, but would answer questions after much persuasion with no evident speech defect. It was difficult to get and hold her attention. There was marked amnesia present. She could not recall her age, where or when she was born, or anything of a similar nature. On account of the psychomotor retardation and apparent dementia the presence of illusions, hallucinations and delusions could not be obtained. There was some emotional disturbance and she would cry when questioned. She was very untidy and slack in appearance, took no interest in things going on about her, and needed very close care and attention.

During the first few months at the hospital she remained in the same apparently demented condition, — semi-mute, untidy and filthy, — although she improved somewhat physically, ate voluntarily and gained in flesh.

At this time there was made a routine Wassermann test of the blood of all of our old cases in the hospital and we found her blood to be positive; accordingly, a Wassermann test of her spinal fluid was made which showed: cells, 4; small lymph, 75 per cent.; large lymph, 25 per cent.; globulin, ++; albumin, +; gold, 555540000.

She was put on mercuric chloride intravenously for a time. A short time after she showed some improvement, both physically and mentally, took better care of herself, talked more and appeared much brighter.

Another Wassermann examination of blood and spinal fluid was made Aug. 1, 1916, both being positive. The fluid gave the gold, 55555432; cells, 23; albumin, +++; globulin, +++; small lymph, 88; large lymph, 8; plasma, 4. She was put on treatment of salicylate of mercury and diarsenol periodically at this time, and the blood and fluid were examined from time to time, always proving positive. The last examination of the fluid, January, 1917, showed gold, 5355555433.

To summarize: this patient showed semi-mutism, apparent dementia, catatonic symptoms, untidiness, amnesia, inactivity and psychomotor retardation, with constant positive serology of paresis, exaggerated reflexes and tremor.

At the present time she shows considerable mental and physical improvement over her condition at the time of her admission, but still with much retardation, with hyperactive knee jerks, very stiff pupils, marked tremor of tongue and fingers and positive Romberg.

Case No. 2. (Mr. J. D.). — Admitted to the Grafton State Hospital Nov. 23, 1915, from another State hospital whose abstract is as follows: admitted there March 27, 1915, male, age 37, single.

Owing to his demented and retarded condition has preserved a state of mutism during greater part of his residence here, and it has been impossible to obtain any information from him in regard to his relatives and friends. He was arrested by the police and admitted to this hospital on the following certificate.

"Onset of attack unknown. He was arrested by police March 24, 1915, for his mental observation. Talked irrelevantly and seemed to be in a moody, depressed mental condition. Says people have not used him right, and talks of suicide. Has torn up furniture of his cell, and on two occasions has attempted to kill himself. No history of drugs or alcohol."

On admission to hospital patient was quiet, orderly, mute and showed decided psychomotor retardation. Throughout his stay in this hospital he has shown a blank, expressionless apathetic countenance, but states he does not feel exceptionally blue. On one occasion showed some ideas of persecution. Has evidently been hallucinated at times. Spoke of seeing faces and various animals, especially at night. He showed partial insight. Patient gradually became much more demented and mute, refusing to answer all questions, and showed very limited mental activity.

Physical examination negative. Wassermann reaction to blood and spinal fluid positive. Urine negative. Pupils react well to light and accommodation. Patient does not react to pin pricks. Knee jerks are diminished equally. Triceps present. Organic reflexes not impaired. Later, however, the patient showed incontinence. There was some swaying in Romberg position with some tremor of extended fingers. Patient was quite restless and resistive at times.

During the remaining time of his residence at this hospital he was mute, was observed staring constantly at the floor, looking neither to the left nor right. He was fairly clean and neat in his personal appearance. He would obey commands slowly, but owing to his retardation and increasing dementia would not speak or co-operate.

On admission to the Grafton State Hospital Nov. 23, 1915, the physical examination was negative other than mitral murmur. Superficial reflexes were present and acute. Knee jerks were sluggish but equal. No clonus or Babinski. Pupils reacted sluggishly to light, and accommodation with consensual absent. His gait was slow, but otherwise showed no peculiarity. He stood steady in Romberg position.

Mental Examination. — He sat with his head resting upon his chest with his hands clasped, paid no attention to questions, and was entirely indifferent to his surroundings. He was extremely feeble in his movements, walked slowly about, was careless about his personal appearance and untidy in his habits. On account of his mutism, illusions, hallucinations, orientation, memory, etc., could not be obtained. Emotionally seemed to be depressed and apathetic. The patient remained in the above condition for a number of months, during which time he was visited by his brother, but would not make any conversation with him and showed utter lack of interest in his visit, but did write the following: "Tell them that I feel different at different times of the day. Sometimes my fingers and muscles are stiff, and headache, and different times my neck gets stiff, crampy like. Tell them that I would like to see them, but it isn't very pleasant when one can't talk to them. Give them my best wishes, that is the best I can do, for I don't feel capable of doing anything rightly, only once in a while for a few moments, or half hour at a time; only my mind moves too fast for my brains, that is as near as I can explain my troubles."

Aug. 14, 1916: Wassermann reaction on blood, positive; on spinal fluid, doubtful: cells, 0; globulin, 3; albumin, 3; gold, 44433321. He continued for the most part mute; would answer questions by "yes" or "no;" would stand for the most part of the day in a particular part of the ward, leaning against the wall.

On Dec. 13, 1916, it was noticed that he had lost the use of right leg. At this time he talked more, and showed fairly good memory and orientation. On December 15 his right arm showed almost complete paralysis. He answered questions quite satisfactorily, and there seemed to be less retardation.

Dec. 19, 1916: The Wassermann test on blood and spinal fluid was positive, the fluid showing albumin, 2+; globulin, 1+; gold, 555555444. At this time he was put on treatment of diarsenol and salicylate mercury, diarsenol being given intravenously and salicylate mercury intramuscularly. The spinal fluid and blood were tested from time to time, the blood alternating between positive and negative. The last test made gave the following blood and spinal fluid negative: cells, 0; gold, positive, but less typical; globulin, 1+; albumin, 2+.

To summarize: this patient showed mutism, lack of interest, untidiness, slow stereotyped movements, apparent dementia, psychomotor retardation, positive serology of paresis, slight tremor, increased reflexes except pupillary, which were sluggish.

At the present time this patient shows remarkable improvement, both mentally and physically. In fact, the improvement has been sufficient to warrant his going away from the hospital on a six months' visit with his brother. He returns to the hospital weekly for his treatments unaccompanied.

While nothing new or remarkable has been advanced by reporting these cases, yet to my mind it shows the importance of doing routine blood examination of both old as well as newly admitted cases, as one of the above reported cases was found by the routine blood examination which would not otherwise have been properly diagnosed, inasmuch as every sign and symptom pointed toward the diagnosis of dementia præcox during the first number of months of its course, and probably would have gone the way of dementia præcox. It goes without saying that the sooner the treatment is started in such cases, before pronounced destructive organic changes occur, the better are the chances of improvement. In light of the foregoing is it not possible that some of our apparently dementia præcox patients who have not had their blood submitted to the Wassermann test may be suffering from some form of neurosyphilis?

AN EPIDEMIC OF DYSENTERY AT THE BOSTON STATE HOSPITAL, DUE TO A MEMBER OF THE PARATYPHOID-ENTERITIDIS GROUP.

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and
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[From the Laboratory of the Boston State Hospital.]

I. Introduction.

The so-called "asylum dysentery" still presents a problem as regards etiology, pathology and prevention. The finding of the Shiga bacillus in connection with numerous outbreaks has led to the assumption that it is always the causative organism. That other organisms may be concerned is demonstrated by the outbreak here reported, which occurred in the infirmary building of the Boston State Hospital in the spring of 1915. The cases were unusual, both clinically and bacteriologically, and therefore it seems of interest to report the epidemic. The severe cases were characterized clinically by acute dysenteric symptoms and the fatal cases by septicemia and bronchopneumonia. The intestinal condition found in the autopsied cases was a wide spread membranous ileocolitis. The cases of moderate severity had blood and mucus in the stools, without much prostration while the mild cases had only diarrhea. The organism isolated in connection with the epidemic is a member of the paratyphoid-enteritidis group. In August, 1915, the disease reappeared in the infirmary, and there was also an outbreak in the other buildings located near the infirmary. Isolated cases occurred in the infirmary during the winter of 1915–16, and in the spring of 1916 small numbers in both the Infirmary and the Cowles building. An effort was made to prevent the occurrence of the disease by prophylactic vaccination. Observations were made on agglutinin formation in the disease and after prophylactic inoculation. Skin tests were carried out with members of the paratyphoid-enteritidis group, and the leukocytic reaction was studied after prophylactic and therapeutic administration of vaccine.

II. EPIDEMIOLOGY.

The infirmary is one of the buildings of the West Group, in which, as is seen by reference to Chart 1, are also the Cowles building for chronically disturbed women, and the Fisher building, containing wards for chronic men patients, officers' quarters, the kitchen supplying the entire West Group, and dining-rooms for the nurses and other employees of the group; and, near the infirmary, the cow and horse barns and the piggery.*

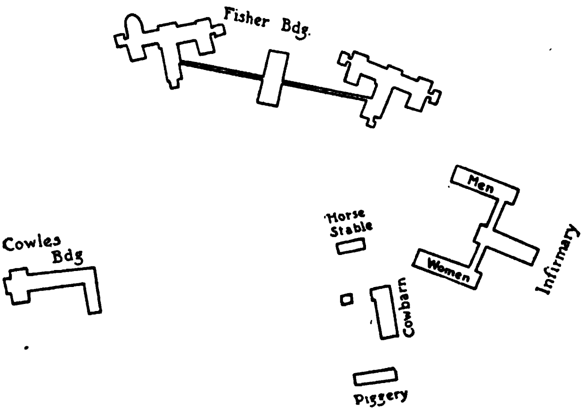

CHART 1.—Buildings of West Group. (Scale of feet: 100 = |——|.)

The first outbreak of the dysentery occurred in the infirmary in April, 1915. In this building are about 380 patients, four wards each for men and women. The class of patients is limited to the elderly infirm and those having a chronic organic disease.

On the women's side the first case appeared April 15 and the last April 30. Eleven patients had the disease in a very severe form, and of these 6 died. One nurse had a short but moderately severe attack, and 3 nurses had diarrhea.

On the men's side the first case developed April 24, the last May 13. There were 8 cases, some quite severe, but none fatal.

The majority of the cases on the women's side came from a ward (V) on which the patients were up and about and some of them very untidy. The other cases, with one exception, appeared on the ward (VIII) to which those having the disease were transferred.

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* During 1916 the piggery was removed to a remote location and cow barns closed.

The first 3 cases occurred within three days' time and varied sufficiently in clinical signs so that they were not immediately classed together. The first appeared as a severe diarrhea in a very untidy senile dement; the second as a dysentery in an untidy paretic; the third patient showed lung signs twenty-four hours preceding the dysentery and died in five days. There was no autopsy. On April 20 the head nurse on Ward VIII, where these patients were isolated, had a diarrhea for one day, beginning twenty-four hours after the transfer of the patients to her ward. On April 25, when 4 more patients on Ward V had a dysentery, 2 nurses had a diarrhea, one of them being a night nurse working between Wards V and IV. On Ward IV a patient was taken ill with dysentery about two days later. As this patient had been cared for especially by the above-mentioned nurse, it is possible that she contracted the disease from the latter. On April 28 the last case on V developed, and on the thirteenth 3 new cases appeared on VIII.

In the earliest cases bacillary dysentery was ruled out by cultures from the stools. The clinical signs were so similar after the disease was once established that there was no doubt of an epidemic. The sequence of cases on the women's side was such that it seemed probable that the disease was being communicated from one person to another; therefore all suspects were at once isolated, and those having the disease were put in single rooms, with strict isolation and careful disinfection. On the suspected Ward V all untidy patients, whether having a diarrhea or not, were put to bed, and their stools inspected and disinfected. No new case developed on the women's side of the infirmary between May 1 and mid-August, the beginning of the second outbreak.

On the men's wards, as on the women's, there was one ward entirely free from the disease. These were both wards on which nearly all the patients were bedridden. On the other wards of the men's side the cases occurred in such sequence that no conclusion could be drawn in regard to the mode of transmission of the disease. A nurse who slept on the women's side, but worked among the men patients, had a diarrhea at the date of the first onset among the men patients, but it could not be satisfactorily proved that she was the carrier. No new case developed on the men's side between mid-May and mid-August.

In the investigation of the source of the epidemic, attention was directed first to the food and milk supplies. The food supplies for the entire West Group are brought over daily from the main storehouse at the East Group, a quarter of a mile distant. The milk comes partly from the hospital herd and partly from a city firm. The food for all persons in the West Group (staff, employees and patients) is prepared in the main kitchen at the Fisher building (see Chart 1) by three cooks, assisted by six patients from that building. The nurses in each ward are assisted in serving the food by several patients living on the ward.

Identification of the organism concerned in the epidemic as a member of the paratyphoid-enteritidis group at once brought the meat under suspicion, in view of the association of bacilli of that type with epidemics of meat poisoning. Cultures were made from the surface and interior of meat and poultry in the West Group refrigerator at the beginning of the epidemic, but the results were negative. Cultures from the milk of both the State Hospital and outside supply were also negative.

Investigation of the West Group kitchen employees and patients, also of the porter and his assistant (patient) who delivered food to the infirmary, elicited no history of diarrhea and they all gave negative agglutination reactions with the epidemic organism. Cases of diarrhea among the patients of the East Group at this time yielded neither the organism nor an agglutination reaction, and at no time since has there been a case of this infection at the East Group. The limitation of the disease to the West Group suggested a contamination of the food after reaching the group. The examination of food and food handlers was made before the two isolated early cases, described in the following paragraph, were discovered.

Finding the agglutination test positive among the nurses who had a diarrhea during the first outbreak brought up the question of possible cases previous to that time. Two such were found: a woman patient who had had a diarrhea for one day only (April 4, 1915), who was not on the ward on which dysentery first developed, and a man patient, who is not recorded as having intestinal symptoms, but who on March 20, 1915, had an indefinite illness characterized by fever and muscular weakness.

A review of death and autopsy reports furnished no clue to earlier cases. There had been no epidemic of any kind at the hospital since a paratyphoid *A* infection in 1910.[1]

The West Group refrigerator was out of use during the first two weeks in April, 1915, while it was undergoing repairs, and during this time the food was kept in makeshift places. The reconstruction of the refrigerator was necessitated because it failed to keep the food cold and was not mouse-proof. For a few weeks before the acute outbreak there had been quite a number of mild diarrheas among the nurses and attendants, also affecting some other persons in the group, but not sufficiently severe to incapacitate them. No attention was paid to these attacks at the time, and only detailed inquiry later elicited a history of them.

A remotely possible complication of the situation is the fact that in the spring of 1914 hog cholera was epidemic among the pigs, and again in August, 1915. Hog cholera is thought to be due to a filterable virus, but associated with the latter are various members of the paratyphoid-enteritidis group. The most frequent of these is B. suipestifer, which was at one time thought to be the cause of the disease. The exact relationship of these organisms to the lesions is still undecided. B. suipestifer is supposedly non-pathogenic for man, but a number of human infections proved to be due to it have been reported. These were of the acute intestinal type. The occurrence of hog cholera the year previous to the human epidemic, the known association of the paratyphoid-enteritidis group with hog cholera, the finding of a member of the group as the cause of the human epidemic, and evidence which tended to show that the latter was not due to a suddenly introduced food infection, raised the question of a possible connection between the animal and the human epidemic. Cultures were made from the lymph nodes and spleen of a hog-cholera animal killed at the hospital in August, 1915. The organism proved to be of the colon bacillus type, and by agglutination reactions did not show a close relationship to the bacillus of the human epidemic.

From a review of the first outbreak it seems probable that the immediate source of the epidemic was not a suddenly introduced food infection; that the disease was communicated from one person to another; that it was endemic, and that previous mild cases of diarrhea among patients and employees had been overlooked. There is a possibility that the cause of these diarrheas may have been contamination of the food in the refrigerator by rats and mice, as these animals frequently harbor bacteria of this group in the intestine (mice, B. suipestifer;

rats, B. enteritidis).[2] It may be possible that these animals were the connecting link between the hog cholera and the human epidemic.

The second outbreak began in mid-July, with mild attacks of diarrhea among the nurses at the Cowles building. These were not mentioned until dysentery appeared among the patients of this building early in August. The Cowles contains about 125 chronically disturbed women, young or in early middle life, and nearly all strong physically. From August 1 to October there were 24 cases, and no ward was exempt.

In mid-August came a recurrence in the infirmary, on both the men's and women's sides, without regard to wards. About the same number of patients had the disease as in the spring, but there was only one fatality. The last group of cases was on the women's side, 1 case on September 30 and 9 between October 23 and 27.

In mid-August and early September there were also a number of cases of diarrhea and dysentery in the Fisher building among patients, nurses and officers.

As to the mode of transmission of the disease in the second outbreak, there seems no doubt but that flies played a leading part, for they were very numerous in all the buildings of the West Group. The buildings are incompletely screened. As breeding-places for flies should be mentioned the stables, barns and piggery, which were situated within a few hundred feet of the buildings for patients.

III. Clinical Description.

The following is a description of the disease as it appeared on the women's service of the infirmary during the first outbreak. The disease as a whole presented such a definite picture that it is unnecessary to describe individual cases. The duration in recovered cases was about three weeks, in fatal cases five to ten days. The incubation period could not be definitely ascertained, but the limits were apparently twenty-four hours and seven days. The onset was characterized by prostration, headache, backache, and large watery stools containing mucus and blood.

The temperature curve was somewhat variable. The initial temperature was between 99° and 104°, but was usually high, remaining so from three to five days, then dropping gradually during one or two days to 99°, and remaining there for about

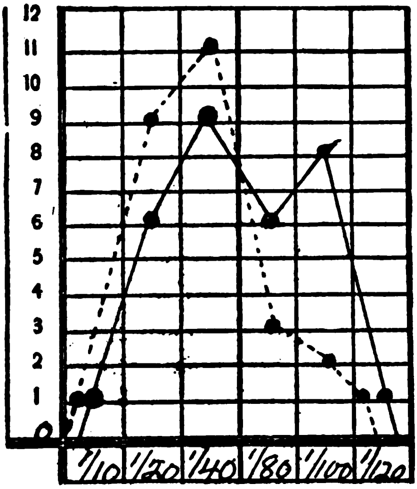

Chart 2.—Agglutination reactions in third and fourth weeks after onset.
(———— Dysentery cases 32; .... Diarrhea cases 27.)

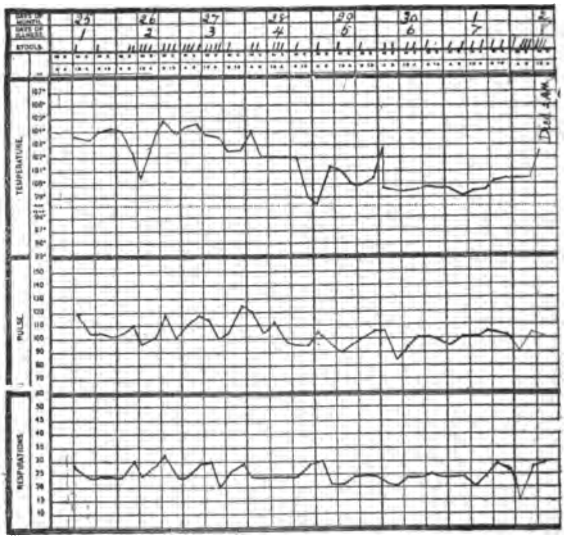

Chart 3.

a week before returning to normal. Occasionally there was a rise to 101° after the fall. The pulse was usually high with the temperature, 110 to 120. One fatal case, however, had an initial pulse of 100 with a temperature of 104°, with a rise in the pulse rate as the temperature fell.

A few hours after the onset the tongue became dry and covered with a white fur and the lips were dry and red. With the fall of temperature the tongue gradually became moist, but the heavy white coat persisted after the dysentery had ceased, even as long as three weeks.

There were no throat symptoms and no glandular enlargement.

The liver and spleen were not enlarged, either at the beginning or as the disease progressed.

Nervous symptoms were not prominent. The headache disappeared within twenty-four hours. Two of the fatal cases became restless, picking at the bedclothes.

The skin showed no rose spots. The face was much flushed during the high temperature, then the face and body became sallow and the skin dry.

The appearance and number of the stools were quite characteristic, six to eight in twenty-four hours, large, watery, brown, containing blood and mucus. The odor was exceedingly foul. In one fatal case, two days before death there was a severe hemorrhage, with large clots of blood. In another fatal case there appeared large pieces of mucus, almost like casts of the intestine. In the severe cases that recovered, the dysentery persisted two to three weeks.

The abdomen was soft and usually not tender. There was some tenesmus for the first few days. In one fatal case the abdomen became distended with gas a few days before death.

There was marked muscular weakness, persisting weeks and even months after recovery from the dysentery. The muscles became very soft and the patients lost weight early.

Clinical Tests. — The urine showed a trace of albumen. The blood picture varied somewhat in different patients and in the same patient during the course of the disease. In no case did an anemia develop. The total white count was rather more variable than the differential, ranging between 5,000 and 10,000, and not decreasing with the progress of the disease. In the severe cases, uncomplicated with pneumonia, the polynuclears were between 54 and 66 per cent during the first two weeks, the lower per cent. for the individual being in the first week. The lymphocytes persisted rather high, between 27 and 40 per cent. Eosinophiles were not found in any case examined during the first week; in the second week they reappeared in .5 to 2 per cent. Transitionals gave a higher per cent. in the first than in the second week. The blood picture of the third week was practically the same as that of the second week, both for protracted cases and convalescents. In the pneumonic-dysenteric cases, the polynuclears were relatively higher, 72 to 77 per cent., but the total count was only moderately increased, 8,000 to 12,000; in the recovered cases of this class the lymphocytes showed a high per cent., 37 to 43 per cent., in the third week. In all cases blood platelets were much increased.

Blood cultures taken in two cases during the first week were negative. A culture from the urine in one very serious case that recovered was negative for the epidemic bacillus, but yielded a streptococcus.

The diagnosis was made from the clinical signs, finding the organism in the stools, and by the agglutination test (see sections on "Bacteriology" and "Agglutination").

Convalescence was slow because of extreme muscular weakness and recurrence of diarrhea with any but the most careful diet.

Sequelæ were not observed, unless the muscular weakness may be regarded as such.

The diet and medical treatment were about the same in each case. Boiled or plain milk and barley water were best taken, and any change from this routine in early convalescence invariably brought back blood or mucus in the stools. At the beginning of the outbreak some days were spent in experimenting with ipecac and morphine and antiseptic or astringent irrigations. The drugs had no effect on the dysentery and the irrigations were irritating. The following treatment was then adopted: a daily high saline irrigation and strychnia sulphur, grams $\frac{1}{60}$ t.i.d. by mouth. Castor oil was used at the onset and for constipation during convalescence.

Vaccine therapy was tried in two protracted cases. The doses were small, beginning with 50,000,000 and increasing 25,000,000 with each dose until four doses had been given, the injection being made every second day. It may have been a coincidence that convalescence began during this treatment.

The mild attacks among the nurses were characterized by a diarrhea lasting about twenty-four hours, with numerous offensive watery movements, accompanied in some instances by griping pains. The diagnosis was made later by agglutination tests.

The clinical description of the second outbreak in the infirmary, from August to October, 1915, corresponds to that of the first, except that the disease was of shorter duration and less severe. The diagnosis of bronchopneumonia was made in two cases about twenty-four hours before the dysentery appeared. Among the patients in the Cowles building the acute stage of dysentery lasted about three days. It was often ushered in with nausea and vomiting, a sharp rise in temperature and increased respiration. The patients were kept in isolation from seven to ten days and recovered with their former physical activity. The cases among the employees were short attacks of diarrhea or dysentery, lasting from one to three days.

An acute arthritis of the knee joint developed in one of the less severe cases ten days after the onset and two or three days after recovery from the intestinal disturbance. The highest temperature during the arthritis was 101°. A week later the other knee became involved, the temperature again rising to 101°. The leukocyte count was 10,000, with polymorphonuclears 71 per cent.

Differential Diagnosis. — The paratyphoid group of infections is usually divided clinically into a gastro-enteric and a typhoid form, to which it appears, from the present observations and those accumulating during the present war, a third form, the dysenteric, should be added. Bainbridge[2] maintains the view that B. gärtneri, B. suipestifer and B. typhi murium, associated with food and especially meat poisoning, cause the gastro-enteric type, while B. paratyphosus *A* and *B*, spread by human carriers, and never found except in connection with human disease, cause the typhoid form. Uhlenhuth and Hübener,[3] on the other hand, consider paratyphoid *A* and *B* also as a cause of acute food poisoning, and state further that the different clinical types may appear in the same epidemic. During the present European war the British have made numerous reports of paratyphoid *A* and *B* infections, describing chiefly the typhoid form, but mentioning also a dysenteric.[4]

The typical gastro-intestinal form comes on suddenly, usually from twelve to forty-eight hours after eating infected food, with headache, chilliness, pain in the abdomen and vomiting, followed by a rise in temperature and pulse, and a diarrhea. The stools are described as exceedingly offensive, and muscular weakness, both during the acute stage and convalescence, is very marked. The acute period lasts from two to five days, and the total duration of the disease is about one week.

The typhoid variety resembles a mild form of true typhoid. Sequelæ are frequent, the most common, perhaps, being an arthritis. Serious local lesions, such as abscess of the lung or osteomyelitis, may follow paratyphoid *B*.

Short descriptions of the dysenteric form have appeared during the war from British and Austrian sources. Bassett-Smith[4] remarks that cases (military) coming home labelled "dysentery" should be regarded as potential paratyphoid, because many of them are found to be that. C. Miller[4] regards the dysenteric as the most severe form of paratyphoid infection seen at the Netley Hospital. The majority, and most of the severe cases, are due to paratyphoid *A*. He describes the patients as emaciated, blue and pulseless, often showing a petechial rash, subcutaneous hemorrhages, and an aphthous stomatitis and pharyngitis. There is constant flux of pink mucus in small quantities, with tenesmus. The mortality is high.

Korczyski[5] describes four cases of paratyphoid dysentery with autopsy. The disease attacked persons who were physically reduced, and toxic features were prominent. He divides the cases into two groups, the enteric, in which the lesions (hemorrhage into mucosa and occasional ulceration) are situated in the small intestine (the stools are feculent, with only traces of blood, and tenesmus is absent); and the dysenteric, in which there is an inflammation of the large intestine, and stools are numerous, small, with much blood and mucus, and there is tenesmus and pain over the descending colon.

The present disease has clinical features of both the typhoid and gastro-enteric varieties, but differs from the usual picture of both. It agrees with the typhoid form in the presence of a septicemia, respiratory symptoms and arthritis as a sequel, but, considered as a whole, does not give at all a typhoid picture. It resembles the gastro-enteric form in that the symptoms, except in the most severe cases, are predominantly intestinal (although in the typical gastro-enteric variety these are usually of the acute diarrheal and not of the dysenteric type), and in the marked muscular weakness. It differs from it, however, in the absence of nervous symptoms and of the features of an acute intoxication. It is conceded, however, that the gastroenteric form may vary in its course, that it may be of long duration, with irregular fever, diarrhea and bronchitis; also that systemic infection may follow the acute stage.

The disease which the ordinary severe case resembles most clinically is, however, neither of the chief forms of paratyphoid infection, but bacillary (Shiga) dysentery. A comparison of the main features of the two diseases is therefore given.

BACILLARY DYSENTERY (SHIGA).	EPIDEMIC DYSENTERY (BOSTON STATE HOSPITAL).
Onset.	
Acute diarrhea and colic.	Acute diarrhea or dysentery. Nausea and vomiting may be first symptoms.
General Symptoms.	
1. Temperature 101°–102° F. Severe cases, 104°.	1. Temperature 99°–103° F. at onset. Slightly irregular course.
2. Headache, general malaise, and, in severe cases, muscular pains.	2. Headache, backache, aching in limbs at onset. Prostration.
Abdominal Symptoms.	
1. Pain, severe colic.	1. Pain early in disease and not severe.
2. Tenesmus, marked.	2. Tenesmus early in disease and not marked.
3. Tenderness on pressure.	3. Tenderness not usual.
4. Abdomen usually somewhat excavated.	4. Abdomen usually soft and normal in appearance. Excavated in 1 fatal case. Distended in 1 fatal case.
Tongue.	
Moist, somewhat coated in slight attacks. Thick, dirty brown coat in typhoid dysentery.	Moist in mild cases and dry in severe cases. Heavy white coat.
Character of Stools.	
1. Diarrhea at onset not characteristic.	1. Large, brown watery stools. Odor very foul.
2. Mucus, small amount at first. Pure mucus and blood within two to three days.	2. Flecks of mucus and blood may appear at onset or after one to two days. Both usually absent in mild cases.
3. Quantity is small; an ounce or less, and stools are frequent.	3. May continue large and watery or become small in amount. About 8 stools in twenty-four hours in both mild and severe cases.
	4. Constipation in convalescence.
Lung Symptoms.	
None.	Respiration increased in nearly all cases at onset. Bronchopneumonia and multiple abscesses of lungs in fatal cases.

Bacillary Dysentery (Shiga).	Epidemic Dysentery (Boston State Hospital).
Bladder Symptoms.	
Tenesmus.	No bladder symptoms.
Incubation Period.	
Two to four days.	Twenty-four hours to seven days.
Duration and Course.	
Light cases, four to eight days. Severe cases, three to six weeks.	Duration of light cases, one to 4 days. Duration of severe or fatal cases, five days to three weeks.
Disease may become chronic.	Recurrences, but chronic cases not observed.
Complications and Sequelæ.	
Parotitis, ascites, peritonitis. Prolapse of rectum frequent.	Acute arthritis, 1 case. Vomiting, 1 case. Prolapse of rectum, 1 case. Muscular weakness, all cases.

IV. Prophylactic Vaccination.

In December, 1915, and January, 1916, prophylactic vaccine treatment was given to 125 women patients in the infirmary. A formalized vaccine was used, prepared from seven strains isolated in the spring and summer of 1915 from the feces of given clinical cases, also from the heart's blood of two autopsied cases. The especially feeble or sick and those who had had the disease were excepted from treatment, and a few other patients refused to be vaccinated. Fifty-one of the stronger and more active patients were given three inoculations, one week apart, the first 500,000,000 and the remaining two 1,000,000,000. A local reaction was usual, but in only a few instances was a constitutional one observed.

Thirty-two patients received two inoculations and 42 only one. The latter patients were among the bedridden, in whom the local reaction was, as a rule, very marked, with redness and swelling of the entire arm. A constitutional reaction, characterized by prostration, also occurred among the majority of this class. The treatment was discontinued among the very feeble patients because of the disturbing effect of the reactions.

From January, 1916, when prophylactic treatment was given, to July, 1916, there were 17 cases of diarrhea among the women patients of the infirmary. The cases were scattered and few in the winter, but appeared in groups on certain wards in the spring. Of these 17 patients, 10 had been vaccinated (8 with three doses and 1 each with one and two doses); 3 had suffered from the disease in 1915, and 4 had had neither the disease nor the inoculations. All attacks, except one, were mild and short. Some of the patients had only a slight rise in temperature, but all were somewhat prostrated. The exception was a fatal case (see case 5, pathological section) of a severe dysentery, in a senile dement who had refused vaccination; but it should be stated that she had not recovered well from a recent pneumonia and was found also at autopsy to have a subdural hemorrhage. Eight of the 17 cases were on Ward V, which was the hotbed of the disease in 1915.

In the Cowles building, also, there occurred in May, 1916, 3 cases of the short dysenteric type in patients who had not previously had the disease.

Although these experiences indicated that neither prophylactic vaccination nor a severe attack gives more than transient immunity, vaccination was continued in July, 1916, all of the Cowles patients and all additional women infirmary patients who were strong enough for the treatment being given three doses.* It was hoped that by vaccinating shortly before flytime, an immunity of sufficient duration might be produced to tide the patients over that season; and that even if the disease could not be entirely eliminated by prophylactic vaccination, at least its type might be reduced from the severe dysenteric to the mild diarrheal.

From July 1 to October 15 there were 6 cases of diarrhea among infirmary patients who had been completely vaccinated, 5 among those incompletely vaccinated, 2 in patients not vaccinated, and 6 recurrences in unvaccinated patients. There was also 1 case of dysentery and several cases of diarrhea among the 125 inoculated Cowles patients.

Tabulation of the results on the women's side of the infirmary gives the following: —

Total patients completely vaccinated from December, 1915, to October, 1916.	64	Cases among these, . .	13	All diarrheas.
Incompletely vaccinated, .	72	Cases among these, . .	8	All diarrheas.
Not vaccinated, . . .	65	Cases among these, . .	7	1 fatal dysentery.
Total women infirmary patients having disease in 1915.	41	Recurrences among these (not vaccinated).	9	1 dysentery (short). Rest diarrhea.

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* The inoculations were made by Mr. John C. Rock of the Harvard Medical School, laboratory interne for the summer of 1916.

The majority of the cases occurring after vaccination and among those not vaccinated are in patients who are feeble yet not strictly bedridden. New bedridden patients do not usually acquire the disease. The recurrences are in patients who at the time of the first attack were feeble and have since become bedridden. They are, in general, much milder than the original attack, and a number were so slight and transient that they would have passed unrecognized if special study were not being given to the subject.

That the new cases and recurrences are due to the epidemic organism is evidenced by the appearance of agglutination reactions in the former and the persistence of reactions at comparatively high dilutions among the latter after attacks.

Agglutinins range usually from $\frac{1}{80}$ to $\frac{1}{160}$ within one month after vaccination and persist at a low level ($\frac{1}{10}$) for at least ten and eleven months (see section on "Agglutination").

It is evident that immunity after this disease is, at least in feeble persons, irregular and probably usually of short duration. In this respect it resembles Shiga dysentery. The duration of immunity after the various forms of paratyphoid infection is not definitely known.

It is clear, also, that as brilliant results cannot be obtained from prophylactic vaccination in this disease as in the case of either typhoid or probably the typhoid form of paratyphoid. The difficulties are two: the first, inherent in the transient character of the immunity; the second, due to the fact that the disease is particularly prone to attack debilitated persons, who as a rule do not stand prophylactic vaccination well. Nevertheless, the writers are convinced that without it cases of the disease would have been more numerous, and, particularly, more severe during the present year, and that it should be continued upon all suitable exposed patients as another way of combating the disease among a class of patients with whom it is difficult to carry out rigorous hygienic measures because of untidiness and lack of co-operation. It is notable that cases after prophylactic vaccination were decidedly fewer at the Cowles building than at the infirmary, this being due, doubtless, to the stronger physical condition of the former patients. The differences found in the leukocytic reactions of strong and weak patients after doses of vaccine (see section on "Leukocytic Reaction") give a further suggestion on this point. It seems probable that prophylactic vaccination among a fairly strong class of individuals is of considerable value, and that results might be much better among the general population or in armies than in insane hospitals.

The writers have been unable to ascertain that until the present war prophylactic inoculation against members of the paratyphoid-enteritidis group has been tried on a large scale, or that it has ever been undertaken under institutional conditions. Vaccination against paratyphoid *A* and *B* is now, of course, being practised extensively in the European and United States armies, in conjunction with antityphoid inoculation, but data on its efficiency will not be available for some time.

Several experiments in vaccination on a large scale against bacillary dysentery have been made, both in European insane hospitals and among the general population. In the former, the results, although not given in detail, are said to have been successful in stopping an epidemic. Shiga[6] in 1898 to 1900 inoculated about 10,000 persons in a district of Japan where epidemic dysentery prevailed most seriously, and diminished the mortality from 20 to 30 per cent. to about zero.

V. SKIN REACTIONS.

Tests for specific skin reactions were tried with a few patients, of whom 12 had had the disease one to twelve months previously; 10 had received prophylactic treatment one to nine months previously; and 7 had had neither the disease nor prophylactic treatment. Tests with paratyphoid *A*, B. suipestifer and B. enteritidis were also made with these same patients. The method was the same as that described by Gay and Force[7] in 1914 for the typhoidin skin reaction, concentrated 5 per cent. glycerine broth cultures being used, with a similarly concentrated glycerine broth solution as a control. The intradermal was preferred to the scratch method as the reaction with the latter was too delicate and transient and often difficult to distinguish from trauma. Different dilutions of the glycerine broth culture were made with normal salt solution (lowest $\frac{1}{2}$, highest $\frac{1}{10}$) with a desire to find what strength would cause a perfectly distinct reaction in the immune and a negative one in the non-immune individual. By the intradermal method a bleb was formed by the injection of .1 cubic centimeter of the dilution, a short needle of a given caliber being used in all cases.

The reaction from the control dilutions was a slight redness of a few millimeters at the point of injection, disappearing in less than twenty-four hours. The reaction from the culture dilutions was an erythema of a diameter, varying with the strength of the dilution, and having, in some cases, a darker red indurated center. The reading was made at the end of twenty-four hours. The indurated area sometimes persisted for a week.

The following observations were made: —

1. The control patients showed reactions that were often difficult or impossible to distinguish from the reaction in those who had the disease.

2. The reaction varied somewhat in intensity in different individuals, but this did not correspond to the time lapsing between the attack or prophylactic treatment and the test, nor was it more marked in those who had had the disease.

3. In 9 cases in which agglutination reactions were made within a few days or three weeks after the skin test, comparisons were made. One patient who had the disease ten months before showed severe local reaction from a low dilution ($\frac{1}{2}$) of the different cultures and a constitutional reaction, with fainting and nausea; three weeks later the agglutination reaction was entirely negative. There were equally marked skin reactions in 2 patients having agglutination reactions of $\frac{1}{10}$ and $\frac{1}{150}$ three weeks later.

4. High dilutions caused a doubtful or negative reaction in certain patients who had had the disease, although causing a distinct reaction in some control cases.

5. Skin tests made with similar broth cultures of other members of the paratyphoid group showed reactions varying slightly in intensity in the different individuals.

6. The concentrated glycerine broth cultures, made up at different times, varied in their results, and apparently deteriorated in a short time, so that any standardization of dilutions was not possible.

These observations would indicate that the intradermal skin reaction with the concentrated glycerine broth cultures has not been of any clinical value in testing the duration of immunity in these cases, or in distinguishing this disease from infections by other members of the paratyphoid group.

Those who have studied the typhoidin skin reaction vary in their conclusions regarding it. Gay[8] considers it of value in indicating the duration of immunity in those having received prophylactic treatment, that revaccination is indicated when the skin reaction is negative. Kolmer and Berge[9] in their study of the typhoidin skin reaction observed that a reaction could be obtained over a longer period in those who had had the disease than in those having had prophylactic treatment only. They found agglutinins and complement-fixing antibodies usually present in those reacting to the skin test. Kolmer and Berge used a powdered typhoidin and control furnished by Gay, but, because of the severe reaction caused by the control, they regarded the reading of the tests as subject to error.

An effort will be made to test these patients with powdered cultures of the same members of the paratyphoid group before setting aside the skin reaction as of no clinical value in this disease.

VI. THE LEUKOCYTIC REACTION.

Gay and Claypole[10] in their experiments in typhoid immunization found that a specific hyperleukocytosis, preceded by a leukopenia, was produced in typhoid immune rabbits by inoculations with typhoid vaccine, and that the important cell in the gain was the polymorphonuclear. The leukocytic response was more rapid and rose to a greater height in the immunized rabbit than in the normal animal. H. I. McWilliams,[11] in a more recent report of similar experiments with rabbits, noted a hyperleukocytosis which was not of a higher grade in the immune than in the non-immune animal.

Both Gay[12] and McWilliams[13] have described the leukocytic reaction following injections of vaccine in typhoid fever. A chill and rise in temperature are coincident with a leukopenia shortly after the inoculation, and a few hours later, with the lowering of the temperature and a lessening of symptoms, there is a high-grade hyperleukocytosis. The relation of the hyperleukocytosis or "leukocytic crisis" to the freeing of antibodies indicates the possible value of these studies in vaccine therapy and prophylactic immunization.

Although our examinations have been limited to human cases and have not the exactness of results obtained in animal experimentation, they have a certain clinical value, because it has been impossible to observe these cases over a number of months.

A polymorphonuclear leukocytosis, both actual and relative, was demonstrated in one protracted case (M. G.) during the first outbreak of dysentery, after a repeated therapeutic dose of the dysenteric vaccine. Six hours after a subcutaneous injection of 75,000,000, the total count rose from 6,400 to 19,400 and the polymorphonuclears from 56 to 76 per cent., with a loss of eosinophiles. No constitutional reaction followed successive small doses in this patient, although there was an apparent improvement in symptoms.

A polymorphonuclear leukocytosis was observed in a second case eighteen hours after an initial prophylactic inoculation of 500,000,000 and six hours after the onset of a severe constitutional reaction, with fever and vomiting. Total count, 18,800; polymorphonuclears, .92; lymphocytes, .08; large mononuclears, transitionals and eosinophiles, 0. This was the only count made in this case. There is no record eleven months later that this patient has had an attack of the disease.

Because of these isolated observations it seemed worth while to study the leukocytic reaction more carefully, and during the seventeen months following the first outbreak several series of examinations were made, including one woman and four men who had neither the disease nor prophylactic treatment, and four women who had had the disease from nine to seventeen months previously. Subcutaneous injections of vaccine were given for two or three successive weeks in doses as in prophylactic treatment, beginning with 500,000,000. In two cases the counts were taken at two-hour intervals during the first twenty-six hours after the inoculation. In all the other cases counts were taken during either the first twelve or the second twelve hours at one-half, one-hour or two-hour intervals.* Differential as well as total counts were made, and a control count was made in each case. There was no constitutional reaction observed in any of these cases and the local reaction was moderate.

The leukocytic reaction consisted of a hyperleukocytosis, with the highest count rising but little above twice that of the normal count for the individual, and a relative increase in polynuclears at one or more periods during the following twenty-four hours, this relative increase not always being synchronous with the highest total count.

The reaction was somewhat more marked after the second or third inoculation than after the first, except in one case, in which the vaccine was injected shortly after a recurrent attack. In this case the reaction was greater after the first injection of 500,000,000, two days after the attack, than after the second inoculation of 1,000,000,000, seven days later. One case (M. G.), referred to above as showing marked reaction to vaccine therapy on the first attack, showed a moderate reaction both in total and differential counts following an inoculation of 500,000,000 nine months later; and on the third day of a recurrent attack, seventeen months later, gave evidence of a reaction only in a relative increase in polymorphonuclears following an injection of 500,000,000. The diarrhea in this last instance ceased a few hours after the inoculation and there was no evident constitutional disturbance.

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* A number of these counts were made by Mr. J. C. Rock of the Harvard Medical School.

There is no record that the individuals chosen as normal cases in this series of examinations have had an attack of dysentery or diarrhea.

In comparing the leukocytic reaction with the clinical history in these cases the following conclusions have been made: —

1. The stronger normal individuals show more reaction to subcutaneous injections of vaccine than the weaker ones who have had the disease and recurrent attacks.
2. The leukocytic reaction for the individual is more marked during or immediately after an attack of the disease and diminishes with recurrent attacks. This corresponds to the clinical observation that the resistance of the individual to the disease is lessened by a previous attack.
3. The fact that there may be a polymorphonuclear reaction to a subcutaneous injection of vaccine, coincident with the cessation of a diarrhea, would indicate the use of vaccine therapy. In the few cases in which this treatment has been given, the results have been favorable for a recovery from an attack.

VII. BACTERIOLOGY.

The method of isolation of the organism was that usually employed for bacteria of intestinal infections. A loopful of the fresh stool was put into a broth tube, portions containing blood or mucus, if present, being selected. After two hours' incubation, surface inoculations were made on Congo red lactose agar plates. The organism predominated on the plates during the acute stage, and was frequently present in almost pure culture. It was found as long as blood and mucus persisted and for some days after the stool began to be formed, but was not isolated after the acute stage, in cases either that recovered or that came to autopsy.

On Congo red plates at the end of twenty-four hours the colonies were round, raised, with smooth edges, translucent gray, averaging ½ millimeter in diameter. During the second twenty-four hours they became an opaque gray, and increased to about 1 millimeter in diameter. They did not form acid.

The organism when first isolated appeared as a small, short, thick Gram-negative bacillus. After artificial cultivation longer forms appeared. When first isolated it appeared nonmotile, but after frequent replants it took on a moderate motility. It grew quickly and well on the usual media.

Cultural Characteristics. — Agar: A raised, moist, gray streak with wavy borders. The growth was delicate at first, but after several replants became heavier and whiter.

Broth: a light diffuse cloud with somewhat ropy sediment.

Gelatin: not liquefied.

Litmus milk: the results were variable.

An acidity, usually slight, developed slowly (within two or three days) and in some cultures remained permanent as long as the tubes were observed (four weeks). Other cultures after an initial acidity became somewhat more alkaline at the end of three to four weeks.

Indol: tested in Dunham's peptone solution at the end of four days. One-half the strains gave a negative, the other half a faint positive reaction.

Fermentation Reactions were tested qualitatively in Hiss' serum water, and quantitatively in sugar-free broth containing 1 per cent. of the substances giving an acidity in the former medium. The substances used were dextrose, lactose, saccharose, maltose, dextrin, salicin, glycerin and mannite.

TABLE I. — *Qualitative Reactions of Epidemic Organism.*

LITMUS MILK.	Indol.	FERMENTATION REACTIONS (LITMUS SERUM WATER).							
		Dextrose.	Lactose.	Saccharose.	Mannite.	Maltose.	Dextrin.	Salicin.	Glycerin.
Acidity, slowly changing to alkalinity in some strains; permanent acidity in others.	0 or faint +	+ gas	0	0	+ gas	0	0	0	0

TABLE II. — *Acidity in One Per Cent. Sugar-Free Broth.*

CULTURE.	Dextrose.	Lactose.	Saccharose.	Mannite.	Maltose.	Salicin.	Glycerin.
M, ...	+3.4	−0.8	Alkalinity	+3.4	+0.2	Alkalinity	+1.3
B, ...	+1.7	−0.4	+1.8	+2.8	+0.1	Alkalinity	+1.9
P, ...	+4.3	−1.0	Alkalinity	+3.2	+0.3	Alkalinity	+0.8
A-15-24 Heart, . . .	+1.9	−0.2	Alkalinity	+2.4	+1.1	Alkalinity	0.0
L, ...	+3.4	−0.9	Alkalinity	+3.2	+1.1	Alkalinity	Alkalinity
W, ...	+2.2	−0.8	Alkalinity	+3.5	0.0	Alkalinity	+0.8
Paratyphoid *A*, . .	+3.1	0.0	Alkalinity	+3.7	0.0	Alkalinity	+1.2
Paratyphoid *B*, . . .	+2.8	0.0	Alkalinity	+4.0	0.0	Alkalinity	+1.5
B. suipestifer, Ark., . .	+2.0	−1.3	Alkalinity	+2.6	+0.3	Alkalinity	Alkalinity
B. suipestifer, Md., . .	+1.9	−1.2	Alkalinity	+3.0	+0.5	Alkalinity	Alkalinity
Paracolon No. 17, . .	+1.5	−0.7	Alkalinity	+2.8	+0.3	Alkalinity	Alkalinity
Paracolon No. 26, . .	+1.7	−0.6	Alkalinity	+3.0	+0.4	Alkalinity	+1.5
B. enteritidis, . . .	+2.0	0.0	Alkalinity	+3.0	0.0	Alkalinity	+1.0

The qualitative results are given in Table I and the quantitative in Table II. The only substances broken down by all strains are dextrose and mannite, the former usually and the latter always with gas production. Saccharose and glycerin were slightly affected in isolated instances. Salicin, dextrin and maltose were not attacked.

A strain of paratyphoid *A* and *B**, two each of B. suipestifer† and "paracolon bacilli,"* and one of B. enteritidis — Gärtner† — were studied for comparison, and the results are included in the quantitative table.

The colon-typhoid group is divided primarily into a lactose-fermenting (B. coli) and a lactose-negative group. The latter is again subdivided according to the action on mannite; no change, B. dysenteriæ Shiga; acid only, B. typhosus, B. dysenteriæ Flexner; acid and gas, paratyphoid-enteritidis group. The members of this last group are negative to saccharose and salicin and must be distinguished one from another by their agglutinative affinities. The present organism falls in a broad way in the paratyphoid-enteritidis group, as it is a lactose-negative bacillus, forming acid and gas in mannite, and not acting on saccharose and salicin. It differs from the generally accepted characters of the group, however, in a number of particulars. A characteristic of the strains isolated in this epidemic is the variability of their action in milk and in the production of indol. Each strain has retained its original characteristics in these respects after more than a year of artificial cultivation. The paratyphoid-enteritidis group is supposed to form no indol, to ferment maltose with gas formation, and paratyphoid *A* to give a permanent acidity in milk, while the other members of the group give an acidity followed by alkalinity.

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* Obtained from the American Museum of Natural History.

† Obtained from the Harvard Medical School. B. suipestifer Arkansas and Maryland.

Recently, however, Krumwiede, Pratt and Kohn[14] have questioned the value of litmus milk as a medium for differentiating paratyphoid *A* from *B*, as in a series of representations of the group they found that the difference was quantitative only, and that there were numerous intermediate degrees of reaction.

The paratyphoid-enteritidis group is admittedly especially complex and unstable, and the question of the identity or mutual relationships of its members is very confusing and is still unsettled. The cultural and chemico-biological characteristics of various members of the group are essentially the same, so that subdivision according to these is impossible. Agglutination reactions,[15] however, while demonstrating the near relationship of members of the group, do permit a further differentiation into a paratyphoid *B* and a "Gärtner-enteritidis" sub-group. Gärtner antisera do not agglutinate paratyphoid *B* bacilli, and conversely B. antisera do not influence Gärtner bacilli. Complement fixation also shows a separation of the group into the same subgroups. Paratyphoid *A* seems to go in the Gärtner, and B. suipestifer in the *B* subdivision. Paratyphoid *B* is quite uniform in cultural and serological reactions, while the Gärtner group is more irregular in both respects.

In addition, other strains have been described which agree in most cultural characteristics with the group, but are not agglutined by paratyphoid *B* or enteritidis antisera. Immune sera for these strains usually agglutinate only their homologous organisms. These may represent transitions between the two subdivisions. Certain of these cultures have been described under the title of paratyphoid *C*.

As to the normal habitat of members of the group: according to Bainbridge[2] true paratyphoid *A* and *B* are never found except in connection with human disease, while the other members of the group are associated with animals. The normal habitat of

B. suipestifer is the intestine of mice and pigs. B. enteritidis is frequently present in the intestine of healthy rats, and this animal is an important source of the organism. The bacillus also causes an infection of cows and calves. The organism has been the cause of frequent epidemics of meat poisoning, the meat either coming from infected animals, or having been contaminated after slaughter, presumably by rats.

VIII. AGGLUTINATIONS.

A series of agglutination reactions was done, using the sera of recovered and vaccinated patients and of immunized rabbits with the epidemic strains, and also with several representatives of the paratyphoid-enteritidis group which had been studied culturally; also the epidemic strains with rabbit antisera for the same representatives of the group.

The points in view were: the extent of agglutinin formation in the disease, its relation to the type of the disease, persistence of agglutinins after recovery and prophylactic vaccination, and the placing of the epidemic organism in the paratyphoid-enteritidis group.

One or more tests were made on 78 persons. The serum of a normal non-exposed person was used as a control.

The reactions were done by the macroscopic method, using formalized cultures as antigens. For the homologous antigen with patients' sera a mixed antigen was used, made of four strains isolated from the feces, two from the heart's blood and one from the intestinal wall of fatal cases.

All the dysentery cases tested gave a positive reaction in dilutions varying from $\frac{1}{10}$ to $\frac{1}{120}$. Twenty-seven cases of diarrhea occurring in patients and employees at the West Group during the outbreaks of dysentery also agglutinated in dilutions varying from $\frac{1}{20}$ to $\frac{1}{100}$.

Agglutinin formation in the disease, as shown by these results, is very moderate. The highest complete reaction obtained was $\frac{1}{120}$. The majority of the sera obtained to within four weeks after onset agglutinated completely only to $\frac{1}{40}$, smaller numbers to $\frac{1}{80}$ and $\frac{1}{100}$, a few only to $\frac{1}{10}$ and $\frac{1}{20}$ (see Chart 2). As to the relation between the type of the disease and the strength of the agglutination reaction, Chart 2 shows that reaction in high dilutions was more frequent among the dysentery than among the diarrhea cases.

Repeated observations on 12 patients who had a single attack (see Table III.) show that there is a rapid drop in the titre during the first few weeks, in some cases a drop to ½ or ⅕ between the third and fourth week, and that agglutinins persist (at a low level) in some cases for a considerable time, — as long as seventeen months in one case and fourteen months in two others, although single negative results appear at five to eleven months.

TABLE III. — *Persistence of Agglutinins after a Single Attack of Dysentery.*

CASE.	Time between Onset and Test.	Highest Complete Reaction.
M. Br.,	3 weeks,	1/40
	10 months,	1/20
	14 months,	1/10[1]
Wm. B.,	3 weeks,	1/80
	5 months,	1/20
	17 months,	1/10[1]
M. Cl.,	3 weeks,	1/80
	4 weeks,	1/40
	5 months,	1/20
Wm. Cu.,	3 weeks,	1/100
	4 weeks,	1/20
	5 months,	1/10[2]
C. F. H.,	3 weeks,	1/100
	4 weeks,	1/80
	10 months,	1/10
	14 months,	1/10
M. Ga.,	3 weeks,	1/100
	5 months,	1/40[3]
R. Pr.,	2 months,	1/40
	6 months,	1/20
Th. S.,	2 weeks,	1/100
	3 weeks,	1/80
	2 months,	1/20
	10 months,	1/20
	14 months,	1/20
Geo. H.,	3 months,	1/20
	11 months,	1/10[1]
M. McD.,	2 weeks,	1/100
	3 weeks,	1/40
	13 months,	1/10

[1] Partial. [2] Negative. [3] Therapeutic vaccine treatment.

TABLE III. — *Persistence of Agglutinins after a Single Attack of Dysentery* — Con.

CASE.	Time between Onset and Test.	Highest Complete Reaction.
M. Don.,	2 weeks,	1/40
	6 months,	1/10
	11 months,	1/10
Ger. W.,	2 weeks,	1/40
	8 months,	1/20
	11 months,	1/10[1]

[1] Negative.

After prophylactic vaccination repeated agglutination tests were made on 10 patients at intervals up to ten months. In the majority of cases the titre within a month after inoculation ranged from 1/80 to 1/160, but in one case was 1/20. Values of 1/20 and 1/10 were usually obtained at five to six months and 1/10 at ten and eleven months.

No extended observations on the persistence of agglutinins after paratyphoid infections are available, although, on the strength of tests on single cases, they are said to disappear rapidly. Dreyer[4] has found agglutinins for *B* are always high, while for *A* they are low, after either the disease or vaccination, as compared with *B* or typhoid.

The following antigens were also tried out with a limited number of patients' sera and with rabbit serum for the epidemic strains: paratyphoid *A* and *B*, B. suipestifer Arkansas, B. enteritidis,[*] and the strain of paratyphoid *A* isolated in the hospital epidemic of 1910.

TABLE IV. — *Sera of Recovered Patients with Different Antigens.*

[± almost complete; ∓ trace.]

PATIENTS' SERA.	Dilutions.	Mixed Strains, Boston State Hospital Epidemic.	Paratyphoid *A*, New Nork.	Paratyphoid *B*, New York.	B. Enteritidis.	B. Suipestifer, Arkansas.	Paratyphoid *A*, Boston State Hospital.
O'B,	1/10	+	+	±	+	+	±
	1/20	+	–	±	+	+	±
	1/40	+	–	±	+	±	±
	1/80	+	–	±	+	±	–
	1/100	±	–	±	+	±	–

[*] For the sources and cultural characteristics of these strains see Tables III. and IV.

TABLE IV. — *Sera of Recovered Patients with Different Antigens* — Con.

PATIENTS' SERA.	Dilutions.	Mixed Strains, Boston State Hospital Epidemic.	Paratyphoid *A*, New York.	Paratyphoid *B*, New York.	B. Enteritidis.	B. Suipestifer, Arkansas.	Paratyphoid *A*, Boston State Hospital.
W, ...	1/10	+	−	−	+	+	∓
	1/20	+	−	−	+	+	∓
	1/40	+	−	−	+	+	∓
	1/80	±	−	−	+	+	∓
	1/100	±	−	−	+	±	∓
McD, ...	1/10	+	±	−	+	±	±
	1/20	±	±	−	+	∓	±
	1/40	±	±	−	−	−	±
	1/80	±	±	−	−	−	±
	1/100	±	±	−	−	−	−
M, ...	1/10	+	∓	−	..	±	−
	1/20	+	−	−	..	∓	−
	1/40	+	−	−	..	∓	−
	1/80	±	−	−	..	∓	−
	1/100	−	−	−	..	−	−
O'M, ...	1/10	+	−	−	..	−	−
	1/20	±	−	−	..	−	−
	1/40	±	−	−	..	−	−
	1/80	±	−	−	..	−	−
	1/100	±	−	−	..	−	−
C, ...	1/10	+	−	±	..	+	∓
	1/20	+	−	±	..	±	∓
	1/40	±	−	−	..	±	∓
	1/80	±	−	−	..	±	∓
	1/100	±	−	−	..	±	∓
S, ...	1/10	+	+	+	..	+	+
	1/20	+	±	+	..	+	±
	1/40	±	±	+	..	+	−
	1/80	±	−	−	..	±	−
	1/100	±	−	−	..	−	−
Normal, ...	1/10	±	−	∓	+	−	∓
	1/20	−	−	∓	+	−	∓
	1/40	−	−	−	−		−
	1/80				−		−

The results with patients' sera, given in Table IV., show, first, the presence of group reactions; second, the absence of strong reactions with paratyphoid *A* and *B*; third, the non-identity of the present organism with the one isolated in the epidemic of 1910; and fourth, high agglutinations of two sera with B. enteritidis, although these results must be discounted to a certain extent by the reaction of the bacillus with normal serum. With stock paratyphoid *A* and *B* sera the epidemic strains reacted scarcely at all (partially at $\frac{1}{10}$).

A rabbit was immunized with five subcutaneous doses of living broth culture of the same epidemic strains used for the mixed antigen. The antiserum (see Table V.) agglutinated the homologous antigen completely at $\frac{1}{500}$, and almost completely at $\frac{1}{1000}$. It reacted with B. enteritidis better than with the homologous strains, giving a complete reaction at $\frac{1}{1000}$. It agglutinated weakly ($\frac{1}{50}$ complete) paratyphoid *A* and *B* and B. suipestifer, but did not affect the Boston State Hospital strain *A*. These results agree in a general way with those obtained from patients' sera.

A strong anti-enteritidis rabbit serum was prepared, which agglutinated the homologous culture completely at $\frac{1}{1500}$ and almost completely at $\frac{1}{2000}$. Curiously, it reacted only slightly (partially at $\frac{1}{50}$ and $\frac{1}{100}$) with the epidemic cultures. It gave a moderate reaction with paratyphoid *B*, but with *A* reacted almost as well as with its own antigen.

A strong anti-suipestifer serum (complete at $\frac{1}{1500}$) also reacted only weakly with the epidemic strains, but as well with B. enteritidis as with its own antigen; it also agglutinated paratyphoid *B* in high dilutions ($\frac{1}{1000}$), but paratyphoid *A* only moderately.

The results of the agglutination tests with both human and rabbit sera illustrate well the group reactions, but further than that are very difficult, or even impossible, to evaluate, in view of the complexity of agglutination reactions within the group. The outstanding result is that the present organism is not an ordinary *A* or *B*. It does not react to any extent, either, with anti-enteritidis or anti-suipestifer serum, although its immune serum agglutinates both organisms. The high reactions with B. enteritidis should probably be discounted to some extent because of the easy agglutinability of the strain.

As to the place of the present organism within the paratyphoid group, it is impossible to say anything more definite than that

TABLE V. — *Rabbit Antisera with Different Antigens.*

[± almost complete; ∓ trace.]

SERA.	Dilutions.	Mixed, Boston State Hospital Epidemic.	B. Enteritidis.	B. Suipestifer.	Paratyphoid *A*, New York.	Paratyphoid *A*, Boston State Hospital.	Paratyphoid *B*, New York.
Rabbit, mixed strains of Boston State Hospital epidemic.	1/10	+	+	+	+	–	+
	1/50	+	+	+	+	–	+
	1/100	+	+	±	±	–	±
	1/200	+	+	±	–	–	±
	1/300	+	+	±	–	–	∓
	1/400	±	+	±	–	–	∓
	1/500	±	+	±	–	–	∓
	1/1000	±	+	–	–	–	–
Rabbit, B. enteritidis,	1/10	–	+	..	+	..	+
	1/50	±	+	..	+	..	+
	1/100	±	+	..	+	..	+
	1/200	–	+	..	+	..	+
	1/300	–	+	..	+	..	±
	1/400	–	+	..	+	..	±
	1/500	–	±	..	+	..	∓
	1/1000	–	±	..	±	..	∓
	1/1500	–	±	..	±	..	∓
	1/2000	–	±	..	–	..	∓
Rabbit, B. suipestifer,	1/10	±	+	+	–	–	+
	1/50	±	+	+	±	–	+
	1/100	±	+	+	±	–	+
	1/200	–	+	+	±	–	+
	1/300	–	+	+	±	–	+
	1/400	–	+	+	±	–	+
	1/500		+	+	±	–	+
	1/1000		+	+	+	–	+
	1/1500		+	+	±	–	–
	1/2000		–	±	–	–	–
Normal rabbit, . .	1/10	–	–	–	–	–	–
	1/50	–	–	–	–	–	–
	1/100	–	–	–	–	–	–

it is not an ordinary *A*, B. enteritidis or suipestifer, but that, according to agglutinative affinities, it resembles the three latter more than *A*. It does not fit well into either of the chief divisions of the group as outlined by Sobernheim and Seligmann.[15] It might probably be considered as one of the *C* types.

IX. PATHOLOGY.

Autopsies were obtained on two cases in the spring epidemic of 1915, also on a patient who recovered from a severe attack and died five months later of other causes, and two cases of the milder form of the disease which prevailed in 1916.

The two acute virulent cases presented similar pathological pictures. The chief conditions found were: first, an acute fibrinous ileitis and colitis, from which an almost pure culture of the organism was obtained; second, a septicemia, the bacillus being isolated in pure culture from the heart's blood; third, cloudy swelling of the liver, with focal necroses in Case 2; and fourth, a pneumonia of the septic type, with miliary abscesses and large areas of necrosis, from which the organism was recovered, associated with streptococci.

The intestine presented a striking appearance. The lesions began a short distance above the ileocæcal valve, involved the entire colon, and, in the first case, the rectum also. The intestinal wall was thickened and covered with irregular patches of dirty yellow-gray exudate, interspersed with bright-red mucosa, frequently showing foci of hemorrhage. The membrane could be scraped off, leaving an eroded hemorrhagic surface. There were no ulcers and only very moderate swelling of Peyer's patches. The regional lymph nodes were enlarged and injected. Microscopically the intestine showed a necrosis extending inward for varying distances, sometimes converting the entire mucosa into a fibrinous membrane. The latter had sloughed in various small areas, exposing the extremely edematous submucosa. The exudate was composed of lymphoid and plasma cells; polymorphonuclears where fibrin was present, and large phagocytic endothelial cells. The latter were present in some sections in considerable numbers but were not as abundant as in the intestinal lesions of typhoid fever.

The pneumonic exudate was characterized by phagocytic cells, similar to those in the intestine but more numerous. Small numbers were also found in the mesenteric lymph nodes, but none in the liver.

Condensed summaries of the protocols are as follows: —

Case 1. — R. K. (A-15-23), aged 50. A much demented case of Korsakow's psychosis, in hospital two years. Died on fifth day of dysentery. Autopsy five hours post mortem. Body well built, well nourished. Body heat and rigor mortis present.

Ventral Section. — Small amount of free fluid in pelvic cavity.

Thorax. — Recent adhesions over posterior portions of each lung. No excess of fluid in pleural cavities.

Heart. — Weight, 300 grams. Valves normal. Myocardium somewhat friable and endocardium gray. The descending branches of both coronaries show occasional plaques.

Lungs. — Right: weight, 295 grams. Posterior portion is markedly congested. Remainder of lung is normal. Left: weight, 570 grams. The upper part of the upper lobe is normal. The inferior portion of the upper lobe and the entire lower lobe are enlarged, deep red, firm and nodular. Cut surface is consolidated, reddish purple, and is beset with dull gray foci ½ millimeter in diameter, sometimes depressed in the center. A large amount of thin purulent fluid can be expressed from the tissue.

Bronchi are deep red and covered with purulent fluid.

Abdomen. — Spleen: weight, 105 grams. Firm, purple-red on section. Malpighian bodies just visible.

Liver. — Weight, 1,450 grams. Capsule normal. Cut surface shows a yellow-brown mottling with indistinct lobulation. Consistence soft.

Gall Bladder. — Distended; no stones or adhesions.

Gastro-intestinal Tract. — Stomach, duodenum, jejunum and upper ileum negative. The lower third of the ileum is thickened and edematous. The mucosa is swollen and red, and is irregularly covered with a granular yellow-gray exudate. From cecum to anus the intestinal wall is edematous, and the mucosa is covered with a shaggy membrane which follows the rugæ. On scraping off the membrane a hemorrhagic and eroded surface presents. No ulcers found.

Lymph Nodes in the vicinity of the cecum are enlarged, soft and grayish pink.

Pancreas shows some injection; otherwise negative.

Kidneys. — Combined weight, 290 grams. Capsules are thickened. Cortex measures from .2 to .6 millimeter; is gray, with areas of injection. Pyramids dark red with a white streak at the bases.

Adrenals show central softening; otherwise not remarkable.

Bladder. — Negative.

Uterus. — Pedunculated myoma, 1 centimeter in diameter on posterior surface.

Retroperitoneal Tissues. — Negative.

Organs of the Neck. — Negative.

Head. — Calvarium, thick. Dura not adherent; is moderately thickened along the longitudinal fissure. Sinuses negative. Pia thickened on inferior surface of cerebellum; not elsewhere.

Brain. — Weight *in toto*, 1,350 grams. Marked atrophy of the convolutions of the frontal, central and parietal regions, most prominent in the frontal fields, and decreasing posteriorly. Consistency of the brain is everywhere increased.

Microscopical Examination. — Lungs: section through one of the pneumonic areas. These are situated around fairly large vessels. At the edges of the lesion the capillaries are distended. In the center of the focus the alveoli are filled with cellular exudate mixed with considerable fibrin and serum, and there are small areas of hemorrhage. The exudate consists of polymorphonuclear leukocytes and large phagocytic cells. The latter often reach large size (five or six times the diameter of a red corpuscle), have a coarse-meshed cytoplasm, and contain red corpuscles, pigment and polymorphonuclears. Three or four of these cells are often present in a single alveolus. In several places the alveoli are filled with solid masses of bacteria. The alveolar partitions are frequently dissolved in the center of the consolidated areas. The large veins show very marked inflammation. Sections through the abscesses show extensive foci of necrosis with masses of bacteria and fibrin in the center, surrounded by a zone of degenerated leukocytes. The bronchioles are intensely affected, the epithelium being absent, the walls infiltrated with fragmented leukocytes and the normal structures obscured.

Heart: Perinuclear pigment prominent; otherwise nothing notable.

Liver: The liver cells are indistinct in outline; their cytoplasm stains a dull granular pink, and the nuclei are pyknotic or stain faintly. Fat content is very moderate. Collections of lymphocytes about the bile ducts are frequent, and the sinusoids are dilated. No necroses or phagocytic cells.

Spleen: Lymphoid tissue is small in amount. Pulp contains much blood and collections of golden brown pigment. No phagocytes.

Gastro-intestinal Tract: Stomach, nothing noteworthy. Intestine, sections from various parts of the lower ileum and the colon show the following picture: at the edges of an affected area there is a superficial necrosis with fibrin network. The underlying blood vessels are engorged, the glands choked with desquamated epithelium and débris and the mucosa thickly infiltrated with polymorphonuclears. The submucosa is extremely edematous and is infiltrated with lymphocytes, plasma and large phagocytic cells, similar to those found in the lung. The vascular endothelium is swollen and the vessel walls infiltrated. Large numbers of bacilli are scattered free throughout the mucosa and submucosa. The muscularis contains clumps of large phagocytic cells situated about the vessels. In more advanced areas the necrosis extends deeply into the mucosa, and the submucosa shows areas of hemorrhage and a moderate infiltration of polymorphonuclears. The vessels in the necrotic areas are frequently filled with thrombi of fibrin and leukocytes. In the most advanced portions the entire mucosa is converted into a membrane of necrotic tissue, fibrin, polymorphonuclear leukocytes and masses of bacteria. The membrane has sloughed in numerous areas, exposing the submucosa. The necrosis and sloughing is irregular in distribution and depth. The lymphoid tissue is not hyperplastic.

Mesenteric Lymph Nodes: No hyperplasia of lymphoid tissue. Large endothelial cells, phagocytic for lymphocytes and red corpuscles are very numerous.

Adrenals: The cortical cells stain well in general and are much vacuolated. Small collections of lymphocytes with a few plasma cells in the reticulosa.

The other organs showed nothing of note. The brain and cord revealed no lesions which could be correlated with the acute infection, and the description of them is, therefore, omitted.

Cultures. — Heart's Blood. Epidemic organism in pure culture.

Consolidated Area of Lung: Epidemic organism plus a few colonies of streptococci.

Wall of Cecum: Epidemic organism in abundance on Congo red plates.

Lymph Node near Cecum: No growth for three days, then scattered colonies of staphylococcus albus and streptococci.

Cerebro-spinal Fluid: Sterile.

Anatomical Diagnosis. — Acute membranous ileitis and colitis, septicemia, septic pneumonia with abscess formation, acute fibrinous pleuritis, purulent bronchitis, cloudy swelling of liver, myoma uteri, cerebral atrophy.

Case 2. — E. H., aged 63 (A-15-24), paranoid dementia præcox. Hospital residence, three years. Died on the eighth day of illness. Autopsy twenty-six hours after death. Body of a somewhat obese woman. Some distention of abdomen.

Ventral Section. — Omentum contains much fat and in its lower third is adherent to the peritoneal wall. Spleen is bound down by adhesions. No free fluid in peritoneal cavity. The intestines are injected, especially the ascending, transverse and descending colon. Subserous hemorrhages are visible in the cecum.

Thorax. — A few light adhesions over the posterior portion of the left base; otherwise the pleural cavities are free and contain no excess of fluid. Pericardial sac contains a slight excess of fluid.

Heart. — Weight, 450 grams. Epicardial fat abundant. Slight thickening of the edges of the tricuspid and the bases of the aortic valves. Myocardium flabby. Right coronary shows a few small plaques.

Lungs. — Right: weight, 550 grams; left: weight, 530 grams. Both lungs show scars at apices. Upper lobes not remarkable. The posterior portion of the left lower lobe is enlarged, bluish red and firm. Tissue sinks in water, and, on section, discloses an evenly consolidated red surface with multiple clusters of yellow dots, 3 millimeters in diameter, arranged around the bronchioles. The right lower lobe is consolidated but does not show miliary abscesses.

Peribronchial Lymph Nodes. — Moderately enlarged.

Spleen. — Weight, 70 grams. Trabeculæ faint; pulp bright red. Malpighian bodies small.

Liver. — Weight, 2,000 grams. Is adherent to the diaphragm. Shape and capsule not remarkable. Cut surface is yellowish red, and normal markings are indistinct. Consistence soft.

Gall Bladder. — Distended; otherwise not notable.

Gastro-intestinal Tract. — Esophagus markedly injected. Stomach contains fecal material. The intestine is negative to within 6 centimeters of the ileocecal valve. The mucosa of the remaining portion of the ileum is thickened, injected and granular. From the ileocecal valve to the splenic flexure the wall is thick and stiff and the mucosa shows areas of membrane and erosion, but these are not as extensive as in Case 1. The descending colon is less involved and the rectum is comparatively free. No swelling of the lymphoid tissue.

Kidneys. — Combined weight, 320 grams. Capsules strip easily. Cortex is 6 to 7 millimeters in depth and is gray, with foci of injection. Glomeruli not visible.

Adrenals. — Markedly softened.

Bladder. — Mucosa is granular and injected at the trigone. Urine cloudy.

Uterus. — Cavity contains a small amount of turbid fluid.

Calvarium. — Of moderate thickness with small amount of diploe. Dura is adherent to inner table. Pia is lifted from the brain surface by a large amount of fluid; is of normal thickness.

Brain. — Weight, *in toto*, 1,440 grams. Convolutional pattern is simple. No atrophy. Consistence is uniformly increased over the dome. Structures at base negative. Basal vessels free from sclerosis.

Microscopical Examination. — The microscopic findings in the lungs and intestine were similar in all particulars to Case 1, and need not be detailed. The liver showed foci of necrosis, and there was also a chronic thyroiditis.

Cultures. — The organism was recovered in pure culture from the heart's blood and urine, and, in large numbers, from the wall of the colon. In the lung it was associated with staphylococcus albus and aureus.

Anatomical Diagnosis. — Acute membranous ileitis and colitis, septicemia, septic pneumonia with miliary abscesses, focal necroses of the liver, acute fibrinous pleuritis, cardiac hypertrophy and dilatation, chronic fibrous endocarditis, mitral and aortic, acute cystitis, subpial edema.

Case 3. — E. McK., aged 76 (A-15-41), showed conditions four months after recovery from a severe attack.

The patient, a senile dement, died of bronchopneumonia, chronic interstitial nephritis and general arteriosclerosis. The intestine, to within a foot of the ileocecal valve, was normal. Throughout the remainder of the bowel were numerous foci of injection in the mucosa, in some places limited to the crests of the folds, in others spread over several centimeters of the mucosa. The mucosa was not thickened, the lymphoid tissue not increased, nor were there signs of healed ulcers. Mesenteric lymph nodes small. The gall bladder contained dark, viscid bile, and the lining was not remarkable.

Microscopically, in the injected areas of the intestine the superficial epithelium was absent, and near the surface of the mucosa were numerous large vacuolated mononuclear cells, occasionally containing the remnants of red corpuscles. The mucosa also showed numerous plasma cells and a few eosinophilic leukocytes. The outer coats of the intestine were normal.

Cultures from the intestinal wall, feces, mesenteric lymph nodes and bile did not yield the epidemic organism.

Cases 4 and 5 show the condition of the intestine a few days after an attack of moderate severity. Both patients had blood and mucus in the stools, and died several days after the height of the attack. In both, also, the dysentery was merely the terminal event in the course of organic conditions fatal in themselves.

Case 4. — (A-16-21) was a bedridden paretic. The mucosa of the lower ileum was slightly thickened and moderately congested. The mucosa of the upper half of the large intestine was uniformly congested but not thickened. In the lower half of the large intestine the wall was moderately thickened and the mucosa dull gray and granular, with patches of congestion but no membrane or loss of substance. The feces in the lower intestine were fluid, gray, slightly frothy, not offensive. Microscopically there was a loss of superficial epithelium and masses of bacteria in the glands, but practically no cellular reaction and no changes below the mucosa.

Case 5. — (A-16-24) was a senile dement who had an advanced chronic myocarditis and an internal hemorrhagic pachymeningitis. The mucosa of the cecum was hemorrhagic, and from there on through the rectum the wall was moderately edematous, showed large areas of congestion, and, in places, also prominent mucous glands. The lesion was most marked in the sigmoid and upper part of the rectum, where the entire mucosa was velvety and dusky. Feces were soft and yellow. Sections showed some slight superficial necrosis, some small localized losses of substance extending into the submucosa, moderate edema of the latter, and considerable swelling of the lymphoid tissue. An occasional phagocytic endothelial cell was found, also moderate numbers of neutrophilic and quite numerous eosinophilic polymorphonuclears.

The organism was isolated in pure culture from the heart's blood of this case.

It is evident from the last two autopsies that in the cases of moderate severity there is nothing striking or characteristic about either the gross or microscopic picture of the intestine, as there is in the virulent cases, but the conditions are such as might be found in any mild inflammation of the bowel.

Pathologically, the acute fatal cases differ obviously from bacillary dysentery in the presence of a septicemia and pneumonia, as well as in the absence of intestinal ulcers. The invasion of the blood stream, particularly, allies the disease with paratyphoid infection. The intestinal lesions, as well as the course of the disease, are more acute and violent than those of bacillary dysentery.

The pathological anatomy of paratyphoid infections is incompletely known, as until recently comparatively few autopsies had been done on either the typhoid or gastro-enteric forms. Since the outbreak of the war autopsy reports have been accumulating. In general, the results are that paratyphoid has more tendency to involve the large bowel than does typhoid; that the lymphoid tissue of the intestine is only moderately affected; that ulcers are frequently absent, and when present are usually scattered and small. The histology of the lesions appears not to have been especially considered. Job and Ballet[16] give the findings in four cases of paratyphoid fever (one *B* and 3 *A*) as intense congestion of the viscera, a moderate degree of inflammation of Peyer's patches without ulceration; swelling, infiltration and congestion of the abdominal lymph glands, and intense inflammation of the spleen, with lymphoid infiltration and hemorrhage.

As to the dysenteric form, Stollkind[4] says that 10 cases with lesions similar to dysentery have been reported. Korczyski[5] found in the enteric form of his cases acute hyperemia of the jejunum and ileum, hemorrhage into the mucosa and occasional small ulcers; in the dysenteric form the same changes, with the addition of an inflammatory process in the large intestine, with thickening of the mucosa and ulcers, which could scarcely be distinguished from Shiga dysentery. He did not find extensive necrosis.

In the gastro-enteric form, the most acute cases may show no microscopic lesion except punctiform hemorrhages through the entire gastro-intestinal tract.[3] In more prolonged cases there may be swelling of the lymphoid tissue, ulceration, and even gangrene of the intestine. The majority of the few reported autopsies on cases dying a week or more after onset have shown hemorrhagic inflammation of the ileum, hyperplasia of the lymphoid tissue, and, frequently, ulcers.

None of these conditions agrees with the virulent form of the present disease, which is an acute extensive membranous inflammation of the lower ileum and the colon without gross ulceration or hyperplasia of the lymphoid tissue, and, combined with septicemia and pneumonia of the septic type, characterized by large areas of necrosis.

The finding of streptococci in the urine of two cases and in the lung of one case, combined with the epidemic organism, suggests that in fatal cases this may be a secondary complication of importance.

As to a possible relation of the disease to hog cholera, we have no crucial evidence, having failed to isolate an identical bacillus from the hog cholera animal. One is struck, however, by certain points of similarity in the pathology of the present disease and that of hog cholera, particularly the simultaneous involvement of the intestines and the lungs. The intestines in hog cholera may present various appearances — from simple catarrh to hemorrhagic or diphtheritic inflammation. The most frequent form of pulmonary involvement is an acute fibrinous necrotic pneumonia. Organisms of the paratyphoid group are most frequently found in the lungs and spleen. Uhlenhuth and Handel[17] bring up the question whether they may not be two diseases included under hog cholera, one, very contagious, caused by an ultra microscopic virus, and a second, slightly contagious, by B. suipestifer.

X. ANIMAL EXPERIMENTATION.

The pathogenicity of the organism was tested on rabbits weighing from 1,800 to 2,000 grams, by both intravenous and subcutaneous inoculation and by feeding experiments.

One-tenth of a cubic centimeter of a twenty-four hour broth culture of a freshly isolated strain given subcutaneously produced no symptoms at the end of three days. There was no local reaction, and the animal was lively and ate well.

Two-tenths of a cubic centimeter of a twenty-four hour broth culture intravenously killed an animal over night. The most striking lesion at autopsy was an extensive necrosis of the liver. Other changes were cloudy swelling of the myocardium and renal epithelium, and diffuse degenerative changes in the cortical nerve cells. The gastro-intestinal tract was negative.

Another rabbit, which in the beginning of the experimentation was given a massive dose of the organism intravenously (.1 cubic centimeter of a moderately thick suspension of a twenty-four-hour agar culture), died over night, showed the above-mentioned lesions in heart, liver, kidneys, thymus and brain, and, in addition, hemorrhage into the small intestine. The intestinal mucosa was thick, velvety, and deep red. The bacillus was isolated from the blood and bile, but not from the hemorrhagic intestinal contents.

A fourth rabbit was fed 1 cubic centimeter of a twenty-four-hour bouillon culture on carrot once a day for three successive days, after which it was fed sparingly on lettuce and carrots once a day. It remained lively, ate well, had no diarrhea, and its temperature was not abnormally high or irregular. It, however, emaciated rapidly and on the eighth day suddenly died. Autopsy showed an atrophic condition of liver and spleen. The mesenteric lymph nodes were enlarged, soft and of a deep red color. The gastro-intestinal tract was not remarkable macroscopically, and the feces were normal. Microscopically, however, there was found at several points in the small and large intestine a necrosis of the superficial epithelium. The surface in these areas was covered with a thick layer of degenerated cells, mingled with masses of bacteria and scattered polymorphonuclear leukocytes. Numerous plasma cells were present in the underlying mucosa, but the remainder of the intestinal wall was negative. There was hemorrhage in the centers of the enlarged lymph nodes; the lymphoid tissue was active; small numbers of mononuclears, phagocytic for lymphocytes, were present, and a large amount of pigment, both intra-cellular and extra-cellular. The liver showed areas of hemorrhage, but no cloudy swelling or necrosis. The spleen contained numerous endothelial cells, phagocytic for pigment. The other organs were not remarkable. Staphylococcus albus was present in pure culture in the heart's blood and the bile. The urine was sterile.

These results show as far as they go that no condition at all resembling the human disease was produced in rabbits by subcutaneous, intravenous or oral administration of the organism. Ordway[18] produced in rabbits a typhoid-like disease by subcutaneous inoculation of another member of this group, B. suipestifer. The constant and most characteristic lesions were necrosis of the liver. The intestinal lesions, which were less constant and striking, consisted in swelling of Peyer's patches and endothelial proliferation.

SUMMARY.

An acute, very contagious disease, of short incubation period, appearing in three forms: the dysentero-septicemic-pneumonic, the pure dysenteric, and the diarrheal. The conditions found in the virulent fatal cases were a membranous ileocolitis, septicemia and a pneumonia with miliary abscesses and large areas of necrosis. The severer forms of the disease occur in debilitated persons. Immunity is of short duration and recurrences (usually mild) are frequent.

The causative organism is a member of the paratyphoid-enteritidis group, more closely allied, as shown by agglutination tests, with paratyphoid *B*, B. suipestifer and B. enteritidis than with *A*, but not identical with any of them.

The disease was limited to certain buildings of the West Group of the Boston State Hospital. The epidemic was preceded by mild diarrheas among patients and attendants, then broke out in violence in one ward, and was passed from person to person by contact. The source of the infection was not definitely determined. The close affinity of the organism with certain members of the group (suipestifer and enteritidis) which are associated with animals suggests the possibility of such an origin. No evidence was obtained connecting the human epidemic with an outbreak of hog cholera which had prevailed in the piggery at the West Group during the previous summer, but the pathological anatomy of the two diseases presents certain features of similarity. The hypothesis is suggested that the infection was introduced by the contamination of food in the West Group refrigerator by rats, the infection appearing first as mild cases of diarrhea, then as an outbreak on a single ward, due probably to gross contamination of the food by a human carrier, the difference in the severity of the disease depending on the physical condition of the persons attacked, and possibly also on the rapid transfer of the virus. The disease has now become endemic and has assumed a mild form.

Prophylactic inoculation will not suppress the disease among feeble patients, although it may have some influence in mitigating its severity. Among strong persons, however, it appears to be of more value in reducing both the number of cases and the type of the disease. Vaccine treatment during the disease has given good results.

A polymorphonuclear hyperleukocytosis occurs after both the prophylactic and therapeutic administration of vaccine. The leukocytic reaction is more marked in the stronger normal individuals than in the feeble ones who have had the disease and recurrent attacks.

Agglutinin formation in the disease is very moderate. Agglutinins for the organism persist in some cases as long as fourteen and seventeen months after the original attack, when there has been no recurrence. After prophylactic vaccination low titres have been obtained up to eleven months.

Intradermal tests with concentrated glycerin broth cultures of members of the paratyphoid-enteritidis group have given unreliable and conflicting results. Tests with powdered cultures are to be undertaken.

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THE TYPE OF FEEBLE-MINDED WHO CAN BE CARED FOR IN THE COMMUNITY.*

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There has been a gradual reaction against the old and harsh methods of dealing with all classes of moral and mental defectives and delinquents. The growth of knowledge in the community of the close relationship of mental diseases, mental deficiency, crime and poverty has displaced the early methods of resort to physical punishment and, later, the method of segregation of the individual; and in their place has grown up a broad sympathy and willingness to study the individual whose reactions are not entirely in accord with the best interests of society.

A few years ago the probation system, of which this State can now be justly proud, was unheard of. During this last year Massachusetts has placed on probation more than 28,000 people; that is, has permitted this number of individuals to lead comparatively normal lives in the community at a great financial saving to the State, to say nothing of the saving in self-respect to the individual, the family and the community, for the present and for the future.

In the treatment of the physically sick hospitals are necessary, but the whole treatment is directed toward returning the individual to the community. In the modern treatment of the insane large hospitals are necessary, yet in this State, under the supervision of the various hospitals, many patients are successfully boarded out with families; also there are large numbers of the insane who recover and resume their normal activities. Then, in Massachusetts there are no State orphan asylums, thus again showing the growth of tolerance in the community towards its unfortunates.

If we were to follow the light of the first flash in the pan ignited by our "eugenic" friends (to whom we are greatly indebted for throwing much light upon the whole problem) it could very plausibly be argued that for the betterment of society any one convicted of crime should never have his freedom. Any

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person who has been adjudged insane should be permanently incarcerated; any person who has been physically seriously ill should not again take his place in society; any child who has been so unfortunate as to lose his parents and thus become a public charge should remain in custody, it being possible in any of these cases that the defective germ plasm of his progenitors, which, perhaps, is the cause of his own misfortune, he himself carries and possibly may transmit to his offspring. But with our growing regard for the individual and belief *in* the individual and his rights, our conscience at once revolts against such extreme measures. When the eugenic light was turned on this problem it would not have been surprising had the social worker in his search for a solution of so many social problems advocated wholesale segregation of unfortunates of all kinds. Instead, he is giving more earnest endeavor to the study of the individual, applying all the tests of modern research in his desire to do what is best for each individual case. It is impossible to study this subject without recognizing that that which is for the best interest of the individual is also for the best interest of the community. Therefore the ground is very safe on which is built a system that has for its first aim the interest of the individual.

There has never been a time when so many activities have been intelligently directed towards the betterment and improvement of the individual who, for any reason, is not reacting normally in the community. When it was determined how large a factor heredity is in the cause of mental defect it was but natural that the community should turn towards the idea of segregation as a great means for the prevention of mental deficiency. The segregation idea is most important, and there is little danger of it being too strongly emphasized. The need for more institutional provision for the care of the feeble-minded in this State is most apparent. Despite the knowledge, however, of the part that heredity plays in the problem of the feeble-minded, yet so strong is the desire in the heart of every parent, guardian, judge, teacher, minister, physician and social worker that every child shall remain in and be a part of the community, that there have not been presented for admission to the institutions any cases of the higher grade types until all means of their remaining in the community have been exhausted. There are, indeed, hundreds of applications for admission on file at our institutions at the present time that show that the only chance for these boys and girls is in the protection and education that an institution affords, as well as for the protection of the community itself.

With the large number of feeble-minded in this State it cannot be considered a practical solution of the problem, however, to segregate all of them in institutions. In fact, such a course, if it were practical from a financial standpoint, would be neither necessary nor desirable. The institution, important as it is, must be considered but a factor in the solution of this problem.

There are large numbers of the feeble-minded in the community, and there always will be large numbers in the community, even after most liberal provision is made. Therefore it seems that the most practical thing to do is to make still more liberal provision for them and aim at a better understanding of how to safely care for the feeble-minded in the community.

In considering what class of feeble-minded individuals may safely remain in the community it is of more importance to study what communities are safe for the feeble-minded. Most communities have certain conditions that make them unsafe for any unsupervised feeble-minded person. Much more depends on the community than on the individual. All feeble-minded persons depend on others for either their proper or improper social reactions. No feeble-minded person has good judgment or can properly adjust himself to his environment. He is dependent on others to properly order his course for him. The majority of the feeble-minded are not vicious but are simply drifters, easily influenced for good or evil. It is therefore impossible to pick out a feeble-minded person and say that he will do well in a community, for his reactions will depend upon the influence to which he is subjected, and the only safe prediction that can be made for his welfare must be based upon knowledge that he will receive proper supervision.

Without special training the feeble-minded person cannot learn to perform the ordinary duties that the normal child naturally picks up, and for this reason we find in the community large numbers of feeble-minded persons who cannot perform satisfactorily the simplest activities. After he has tried this job and that, only to find that he is a failure, is it any wonder that he becomes a serious social problem? How many normal people could continue to react normally in the community if they were robbed of the great opportunity of industry?

With the spread of knowledge of the feeble-minded problem there has been a great demand for increased provision by the school systems for special classes. These classes are becoming powerful factors in the solution of this problem. The teachers for these classes are selected for superior ability. Their remuneration is greater than that of teachers of the ordinary grades, thus giving permanency to their work, making it possible for a teacher to have the same group of children under observation for a period of years. These classes, all under the general supervision of a trained supervisor, and under medical supervision that is provided, make a most excellent organization for caring for the defective children in the community. They are giving these children the advantage of an education such as is provided in an institution, with the advantage also of receiving home care. They are also extending their activities, in some degree, beyond the school by assisting these boys and girls in securing positions, and in some cases carrying supervision to them in their work. These classes also provide a splendid opportunity for the study of the social reactions of these children in the community, a side of the work that it is impossible for the institution to perform. The supervisor of these classes thus has the opportunity to arrive at a fair conclusion as to what children should have institutional care and what children have fair chances for leading harmless and, possibly, useful lives in the community.

With the extension of this movement for special classes, until every school system of any size has a sufficient number to accommodate all children with mental defect, it would seem that the larger number of children with ordinary mental defect could be safely protected and educated in the community. It is, however, with the advent of mature life that the more difficult problems of dealing with the feeble-minded in the community arise. The adult feeble-minded are more difficult to control than the children, due to the more complex social relations into which they are thrown, but chiefly to the failure of the community to disassociate age and responsibility and make proper provision for the supervision of the irresponsible.

This State has already taken a census of its feeble-minded. Would it not be desirable for the Commission on Mental Diseases to keep a permanent registry of all the feeble-minded in the State and work out a system whereby it can place them all under intelligent, co-ordinating supervision? It would be necessary to have a corps of trained workers selected, preferably from institutional employees and teachers of the special classes, — a group of workers who have lived with, worked with, and who know the feeble-minded. These workers could follow defective children leaving the special classes and give them a kindly oversight and supervision that will largely insure against their becoming harmful agents in the community. The feeble-minded most in need of institutional care could better be determined. The institutions could also perform a larger service by co-ordinating with the community in the way of admitting those most in need of institutional care, and placing out in the community many trained cases, were it possible to place them under the supervision of trained workers, those who know the danger signals of the feeble-minded and who have authority to remand to the institutions.

At the present time we are touching the problem only in spots. We are training and caring for the children in the special classes to a certain age and then turning them out into the community — large numbers to become social problems because they have no longer intelligent supervision. There are already hundreds of social problems of the feeble-minded who need institutional care, but for whom there is no provision. There is no doubt that many of these problems could be prevented if there were room in the institutions where some of the children leaving the special classes could be placed for a time, and thus complement the work of the special classes by tiding these children over, perhaps, an especially critical period in their lives; and while doing this, teaching them a useful occupation and placing them later in the community, still carrying to them the supervision they need.

The activities of the various organizations are based upon the expectation of normal reactions by the individual aided. Therefore too often are their agents applying the same methods with the feeble-minded person that they use so successfully in dealing with the normal person, and, consequently, expecting the same results only to be disappointed time after time with the results obtained. The feeble-minded appear as a foreign body and a check under the wheels of their activities.

If it is necessary to make special provision and select a special group of teachers to supervise and educate the feeble-minded children, it is even more essential to make special provision for the supervision of the feeble-minded adult population in the community.

The feeble-minded problem, although closely associated with the various social problems that the organized societies are dealing with, is still a distinct and separate problem that can be best handled by those who are trained with the feeble-minded.

If the authority of the central commission were increased so as to take under a trained supervision all the feeble-minded in the community, it would have the effect upon the various organizations of liberating a tremendous amount of their energy that is now consumed by the feeble-minded problem.

It would be quite possible to indicate classes of the feeble-minded that we think would safely get along in the community, such as young children, the lower grade cases if provided with physical comfort and care, the male, perhaps, more safely than the female, the girl who is not the possessor of physical beauty, the child who has a good home and others, but this would all be but a guess on individual cases.

In closing I can only say that the number of feeble-minded that can safely be cared for in the community is in direct ratio to the supervision that the community is willing to provide, and I believe this could be most easily effected through the extension of the authority and activities of our Commission on Mental Diseases.